ARCHIVES OF OTOLARYNGOLOGY

VOLUME 21 APRIL 1935 NUMBER 4

ABSCESS OF THE BRAIN

REPORT OF FIVE CONSECUTIVE RECOVERIES, WITH SPECIAL REFERENCE TO THE MOSHER DRAIN AND PNEUMOGRAPHIC VISUALIZATION OF THE ABSCESS CAVITY

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If one allows for the highly specialized response of cerebral tissue to infection, the treatment of abscess of the brain, like the treatment of abscess elsewhere in the body, resolves itself into two major considerations: (1) What is the best time for operation, and (2) what method of drainage gives the best results? These two problems have been fully discussed by Frazier [1] in a recent communication.

There seems to be little doubt that the optimum time to drain an abscess of the brain is after encapsulation has taken place. Grant [2] in a review of fifty-one cases of abscess of the brain has strikingly brought forth the unfortunate results of hasty intervention. Of nineteen patients on whom drainage of the abscess was attempted before the lapse of the six week period from the onset of cerebral symptoms not one survived. Encapsulation of a suppurative focus in the brain rarely takes place in less than six weeks from the initial cerebral complaint, and if the operative manipulations are withheld until after this period and then confined within the limits of the capsule wall a good result should be obtained in most instances. Only in exceptional cases is an emergency operation necessary. It is far better, whenever possible, to delay surgical intervention until the surrounding cerebritis has subsided and a firm capsule has formed about the area of suppuration. Woltman [3] has shown that when the spinal fluid cell count gives evidence of a fall in the total number of cells in association with a predominance of lymphocytes over polymorphonuclear cells this may be taken as an indication that the infection is subsiding and that encapsulation is progressing satisfactorily.

From the Neurosurgical Services of the Brooklyn Jewish Hospital and the Mount Sinai Hospital.

1. Frazier, C.: How Shall We Treat Brain Abscess? Surg., Gynec. & Obst. **57**:122 (July) 1933.

2. Grant, F. C.: Concerning the Mortality from Abscess of the Brain, Arch. Neurol. & Psychiat. **28**:1443 (Dec.) 1932.

3. Woltman, H. W.: Spinal Fluid Cell Count and Encapsulation of Brain Abscess: Attempt to Correlate These Factors and to Determine the Optimal Time for Drainage, J. A. M. A. **100**:720 (March 11) 1933.

As to the best method of draining an abscess of the brain there is still considerable difference of opinion. For this reason it appeared sufficiently important to report five cases of cerebral abscess in which recovery took place after treatment by a reasonably constant procedure. From the experiences gained in these cases it is fair to make only a few generalizations. The Mosher [4] drain was used in every case. In addition, pneumographic visualization of the abscess cavity proved to be of great value in two cases.

In the order of their importance, the following factors seem to have been responsible for the recovery of the five patients: (1) the evacuation of the cavity of the cerebral abscess after encapsulation had taken place; (2) the proper sealing off of the subarachnoid space with a

Fig. 1.—The Mosher drain.

coagulating current to prevent meningitis; (3) the use of the Mosher drain which establishes adequate drainage without repeated manipulation; (4) the accurate visualization of the size, shape and location of the abscess; (5) the adoption of Dr. King's [5] principle of allowing the wall of the cerebral abscess to herniate; (6) the initial and complete removal of the primary focus of infection.

A description of the Mosher drain, which was used in each case for varying periods, is of interest. The use of this drain of copper wire gauze (fig. 1) was first reported on by Mosher in 1916 after gratifying

4. Mosher, H. P.: The Wire Gauze Brain Drain, Surg., Gynec. & Obst. **23**:740 (Dec.) 1916.

5. King, J. E.: The Treatment of Brain Abscess by Unroofing and Temporary Herniation of Abscess Cavity with the Avoidance of Usual Drainage Methods, Surg., Gynec. & Obst. **39**:554 (Nov.) 1924.

results had been obtained in three successive cases. It is a modification of the British army drain which proved satisfactory during the World War in the treatment of suppurative infections of the brain. The method of using the Mosher drain may be briefly summarized as follows: After a generous exposure of dura is made over the site of the abscess, the dura and subarachnoid space are sealed off at the osseous margin with an electric coagulating machine. A needle is then introduced into the abscess, and when pus begins to escape the stylet of the needle is quickly replaced. A cone of dura, underlying brain and abscess wall is then removed with the electric scalpel. The drain, which has been previously lubricated, is inserted into the cavity of the abscess, the obturator removed and the pus allowed to escape freely.

REPORT OF CASES

CASE 1.—*Chronic otitis media of eight years' duration; mastoidectomy disclosing an acute process superimposed on chronic mastoiditis with destruction of the tegmen and a cholesteatoma. Subsequent nocturnal headache, vomiting, hemiparesis and coma. Drainage of abscess of the right temporal lobe with the Mosher drain. Complete recovery.*

On Aug. 5, 1933, an Italian shoe worker, aged 39, came to the Brooklyn Jewish Hospital complaining of severe frontal headache of one month's duration. Since the age of 8 years he had had a purulent discharge from the right ear.

Except for the discharge he had been in good health until one month before admission, when he started to have attacks of severe frontal and vertical headache. At times the headache was associated with pains radiating to the right side of the face and teeth. One morning shortly after the onset of the headache, while on his way to work, he was suddenly seized with a violent spell of dizziness which caused him to return home. Nausea and vomiting occurred frequently during the following two weeks, and there was notable failing in vision.

On admission he was mentally sluggish, spoke deliberately and yawned frequently. He complained bitterly of frontal headache. The temperature was 99 F. and the pulse rate 60. Tbe positive findings were: (1) fulness of the vessels in the right fundus, (2) purulent discharge from the right ear, (3) some stiffness of the neck and (4) bilateral Kernig and Brudzinski signs.

Laboratory studies revealed leukocytosis, the count being 12,300, with 80 per cent neutrophils. The spinal fluid was under 380 mm. pressure and contained 160 cells per cubic millimeter, all polymorphonuclear; the sugar content was 57 mg. per hundred cubic centimeters, and the total protein, 28 mg. There were no organisms on smear or culture. Roentgenograms showed a sclerosing process of the right mastoid.

Four days after the patient's admission there was definite tenderness over the mastoid. A radical mastoidectomy, performed by Dr. Frederick Stone, disclosed an acute process superimposed on an old chronic mastoiditis with destruction of the tegmen. There was a small cholesteatoma of the antrum and attic, and an epidural abscess was present in the middle fossa with a localized pachymeningitis externa. The lateral sinus was normal. The squama was removed until normal dura came into view. No fistulous tract could be identified.

On the following day the patient was still drowsy; he complained of frontotemporal headache, but there was less nuchal rigidity. The pulse rate varied from

50 to 85 per minute, and the temperature ranged from 99 to 100 F. Within the following few days the headache subsided, but there was definite papilledema on the right. On the sixth postoperative day, for no apparent cause, the patient became excitable, could not be reasoned with and insisted on leaving the hospital against advice.

He returned to the hospital six days later. During the interval he had nocturnal headache and was extremely restless. For two days before readmission he vomited repeatedly. On the morning of entry he became drowsy, by afternoon he was stuporous, and in the evening, in a comatose state, he was hurriedly brought back to the hospital.

The positive neurologic findings at that time were: rigidity of the neck; dilatation of the pupils, the right pupil being larger than the left and both reacting sluggishly to light; weakness on the left side of the face; flaccid left hemiplegia; absence of abdominal responses; bilateral Kernig and Babinski signs, and ankle clonus on the right.

The spinal fluid was clear; there were only 10 cells per cubic millimeter, and no organisms were found on smear or culture. The temperature was 99 F., the pulse rate 75 and the respiratory rate 15.

Operation was performed that night. An incision 6 inches (15.24 cm.) long was made from the mastoidectomy wound in an upward and medial direction, and sufficient bone was removed to expose an area of dura about the size of a half-dollar. The dura appeared healthy, and the edge adjoining the bone was coagulated with an electric coagulating machine. A ventricle needle was inserted into the temporal lobe, and pus was obtained at a depth of 3 cm. The stylet was replaced, and a cone of dura, brain and abscess wall was removed in one with the electric scalpel; then the Mosher drain was inserted. The patient almost instantly regained consciousness and began to move the left arm and leg. The postoperative course was smooth; the drain was spontaneously extruded on the fourteenth postoperative day. The patient was discharged on September 9, thirty-five days after the operation, at which time he had been out of bed with no abnormal signs or symptoms for two weeks. The period from the onset of headache to the time of operation was forty-five days.

At the time of writing the patient has been back at his work, working forty hours a week, for seventeen months, and has been in excellent health.

Comment.—One could hardly hope for a more satisfactory sequence of events. The operation was performed just after the six week period; the abscess was encountered on the first attempt; the patient regained consciousness immediately after the operation, and shortly thereafter there was complete return of motor power. After a fortnight the Mosher drain was extruded spontaneously, and since then repeated neurologic studies have failed to disclose the slightest abnormality.

CASE 2.—*Chronic bilateral otitis media since childhood; acute exacerbation of mastoiditis on the left with meningeal involvement. Operation showing acute mastoiditis and a large cholesteatoma. Ten days later drowsiness, aphasia and right hemiparesis. Drainage of abscess of the left temporal lobe with the Mosher drain. Recovery complete except for a residual defect of the visual field.*

A housewife, aged 32, was admitted to the Brooklyn Jewish Hospital on Sept. 11, 1933, because of pain in the left ear of eight days' duration and headache and vomiting for the preceding two days.

When the patient was aged 4, after an attack of scarlet fever and diphtheria, there developed a chronic discharge from both ears with bilateral progressive deafness. The deafness led to some lagging of speech and retardation at school. Though at the time of admission the patient could hear when spoken to loudly, she understood the spoken word best by lip reading. Her relatives stated that her hearing and speech were about the same as before the onset of the recent illness.

The patient seemed perfectly well until three weeks before admission, when a profuse discharge from both ears was noted. Two weeks later she complained of pain in the left ear; at that time her physician found a furuncle in the canal and treated it conservatively. Within the next two days the pain in the left ear recurred, and there was an elevation of temperature from 101 to 102 F. Two days before admission the patient began to have headache on the left side radiating to the occipital region. The temperature rose to 104 F., and vomiting occurred once during the night before admission.

Examination revealed an acutely ill woman, rather stuporous, with a slight foul-smelling discharge from the left ear. The left aural canal was filled with granulations, and on the right some granulations were found protruding from an old perforation in the drum. There was tenderness over the left mastoid; the tonsils and pharynx were injected; the temperature was 103.8 F., and the pulse rate was 110. The patient showed marked nuchal rigidity, a bilateral Kernig sign and hypo-active reflexes. The fundi were normal. Roentgenograms of both mastoids gave evidence of marked sclerosis, but on the left there was a punched-out area overlying the anterior sinus wall; this was interpreted as possibly indicating a chronic abscess or cholesteatoma.

Laboratory studies revealed leukocytosis, the white cells numbering 27,000, with 96 per cent neutrophils. The spinal fluid contained 5,800 cells per cubic millimeter, with 99 per cent polymorphonuclears; the sugar content was 41 mg. per hundred cubic centimeters, and the total protein, 52 mg. The patient was given 20 cc. of antimeningococcus serum intravenously and an equal quantity of serum intraspinally. Smear and culture of the spinal fluid showed no organisms.

A radical mastoidectomy on the left was performed by Dr. Frederick Stone, and a large cholesteatoma was removed from the antrum and middle ear. The middle fossa was exposed until normal dura was visible. A localized area of purulent exudate was found on the dura in the region above the antrum; on culture this was reported to contain Staphylococcus aureus. A part of the squama and the entire tegmen were removed. The lateral sinus was exposed and found to be normal.

The following day the patient was more alert; the temperature was not as high, and the neck was less rigid. She improved steadily for six days and then became restless and more apathetic. The stiffness of the neck and the Kernig sign returned, but the fundi remained normal. The spinal fluid at that time contained 2,600 cells per cubic millimeter, all polymorphonuclears; the sugar content was 50 mg. per hundred cubic centimeters, and no organisms were found on smear or culture.

One week after the mastoidectomy a rash due to the serum developed and was associated with fever, drowsiness and increased stiffness of the neck. The following day the right side showed facial weakness, paresis, hypesthesia and exaggeration of the deep reflexes. The patient was definitely aphasic.

Within the following two days the condition became worse. There was no abnormality in either fundus, though the response to gross tests showed a distinct right homonymous hemianopia. The aphasia and hemiparesis became more pro-

nounced; abdominal responses could not be elicited; the Oppenheim sign was present on the right, but there was no Babinski sign. Drowsiness steadily increased. The spinal fluid at this time contained 110 cells per cubic millimeter with 85 per cent lymphocytes. Smears and culture of the fluid failed to show any organisms. On September 22 a procedure similar to that described in case 1 was performed, except that the bony defect was made more anteriorly.

For the next three or four days the condition did not improve, but there was no further discharge from the drain. Apparently the abscess had completely emptied itself. It was thought best to remove the basket and allow the exposed brain to herniate, according to Dr. King's method. Within the next two days the patient was more alert, though aphasia and hemiplegia were still evident. On the sixth postoperative day the patient began to speak words and to move her right leg. Improvement was steady in all respects, but on the twenty-third postoperative day papilledema of 2 diopters was noted on the left and beginning papilledema on the right. During the following two days the papilledema on the left

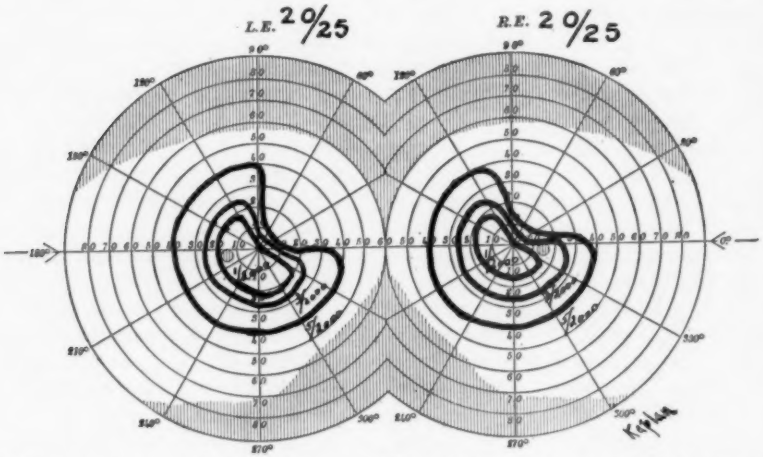

Fig. 2 (case 2).—Visual field on April 15, 1934, showing a residual homonymous defect in the upper right quadrant.

advanced, showing minute hemorrhages and some exudate, and the right disk became completely obscured.

These findings were suggestive of one of two conditions, a recurrent abscess of the brain or a localized basilar meningitis. However, the improved condition of the wound and the favorable clinical appearance of the patient were such that the more benign lesion of localized meningeal inflammation was believed to exist. There was no change in either fundus for the next three days, and then the papilledema began to recede.

The patient was discharged from the hospital on October 25, thirty-three days after operation; at that time the fundi were normal; the power of speech was practically as before the onset of the illness, and there was no motor, sensory or reflex abnormality. She has remained well. The only complaint has been intermittent difficulty in seeing things on the right side. The defect of the visual field has improved and the patient has learned to accommodate for the homonymous defect in the upper right quadrant (fig. 2).

Comment.—The unusual feature in this case was the complete emptying of the abscess with no clinical improvement. The removal of the drain allowed moderate herniation of the edematous brain which apparently was sufficient to restore consciousness. With the recession of the cerebral tissue, motor function and speech returned. The appearance on the twenty-third postoperative day of papilledema which subsided within the following week undoubtedly is best explained on the theory of a temporary interference with drainage in the cisterns of the middle fossa. The defect of the visual field (fig. 2) is the only neurologic abnormality resulting from the operation.

CASE 3.—*Acute suppurative frontal sinusitis complicated by extradural abscess and osteomyelitis of the frontal bone. Headache, papilledema and jacksonian seizures on the left side. Ventriculography. Subtemporal decompression with improvement. Return of headache, followed by coma and sterile meningitis. Removal of the right frontal bone for osteomyelitis; drainage of the abscess in the precentral area with the Mosher drain. Recurrence of abscess; pneumographic visualization and drainage. Tonsillectomy; second recurrence of the abscess; repetition of pneumographic studies; drainage using King's principle. Recovery with residual hemiparesis.*

The history of this patient can be given only in brief. He was under treatment for seventeen months and underwent eleven major operations, eight of which were intracranial procedures. Though the physiologic result leaves much to be desired, there is some satisfaction in having a live patient with an acute and active mind, who in the near future will be self-supporting.

A jeweler, aged 21, entered the Brooklyn Jewish Hospital on May 6, 1933. He stated that he was perfectly well until ten days before when some coryza and cough developed. He soon began to experience frontal headache on the right side; this became increasingly severe. Three days before admission the right upper eyelid became red and sufficiently swollen to close the eye. There were no chills, vomiting or convulsions.

On examination the patient appeared acutely ill; he complained of severe frontal headache; the right upper eyelid was red, hot, edematous and so tense and painful that the eye could not be opened. The left upper lid was also red and edematous, but to a much lesser degree. The right side of the forehead exhibited some edema and exquisite tenderness; there was only slight tenderness over the left frontal region. The pharynx was congested, and there was an accompanying postnasal drip. The temperature was 105 F., the pulse rate 95, the respiratory rate 25 and the blood pressure 124 systolic and 68 diastolic.

Laboratory studies showed leukocytosis, the white cells numbering 15,000 with 84 per cent polymorphonuclears and 16 per cent lymphocytes.

On the following day a radical frontal operation was performed by Dr. Charles Breitman. Through a curvilinear incision on the right the periosteum was elevated over the right frontal and ethmoid regions. Considerable pus was found in the orbital space. The anterior wall of the frontal sinus, the lacrimal bone and the nasal process were removed. The frontal and ethmoid cells were found to be filled with pus. The middle turbinate was removed, and rubber tubing with two strips of iodoform gauze was inserted into the sinus through the nose; the wound was closed in layers, and a rubber drain was left in the subcutaneous tissue. Culture of the purulent material was reported to show Staphylococcus albus-haemolyticus.

The following day the patient complained of diplopia; the temperature dropped to 103 F., and improvement was apparent. The condition continued to improve, and two weeks after admission the patient was discharged.

Six days later he was readmitted. He stated that on the day after his discharge he had excruciating headache on the right side, fever and swelling and pain over the right eye. This was soon followed by some weakness on the left side of the body. He vomited twice during this interval.

The patient did not appear acutely ill, though he complained of some frontal headache. The right upper eyelid was red and edematous, and there was tenderness over the right frontal sinus. The nasal margins of both disks were blurred; the vessels were full and tortuous; in short, there was beginning papilledema. With the flattening of the left side of the face there was also paresis of the left arm. Nuchal rigidity was slight, but the Kernig sign was definitely present on both sides. Abdominal responses could not be elicited. The left biceps was more active than the right, and the Babinski sign was present on both sides.

The spinal fluid was clear and the pressure was normal; the fluid showed no cells, but the presence of sugar was evident.

Dr. Philip Leibowitz performed an operation for reexploration of the right frontal sinus and removal of the posterior wall. As the right frontal lobe was exposed for a distance of 6 inches (15.24 cm.), an epidural pocket containing 10 cc. of pus was encountered and drained. The abscess extended from a recess in the frontal sinus backward about ½ inch (1.2 cm.) to the right of the midline. The dura over the frontal lobe was thickened and covered with red granulations. In the middle of this exposed region a drop of pus was apparently making its way to the surface from beneath the dura. It seemed wise not to explore further at this time; a rubber tissue drain leading from the granulating surface of the dura to the outside was secured, and the wound was closed.

The neurologic findings were unaffected by the operation; the pulse rate remained lower than one expected with the fever. The headache persisted, and the paresis of the left arm became more marked.

It was soon noted that in addition to the left hemiparesis there were also left hemihypesthesia and left homonymous hemianopia. The deep reflexes were more active on the left, and the Babinski sign was present on both sides.

Two weeks after the extradural exploration the wound was again investigated, and more diseased bone was removed. Exploration of the right frontal lobe with needles inserted in all directions failed to reveal a collection of pus.

For the following fortnight except for some elevation of the disks there was no change, but shortly thereafter the patient had his first convulsion. This was a clonic seizure, which lasted four minutes; it started in the left side of the face, spread to the left arm and then became generalized. Eleven days later five such seizures occurred within two hours with resulting increase in the symptoms on the left side, pain over both eyes and severe headache on the right. The spinal fluid was under markedly increased pressure but showed no cells.

Intravenous administration of dextrose and repeated lumbar punctures brought about some relief of the headache and a lessening of the convulsions. Encephalograms were unsatisfactory. Papilledema in both eyes advanced. Ventriculograms made on July 19 (fig. 3) showed a shift of both ventricles to the left and narrowing as well as flattening of the body of the right ventricle. In the hope of preserving the patient's vision and relieving the headache a subtemporal decompression on the right side was performed. One month after the subtemporal decompression the patient was discharged free from headache, fever and convulsions but with a remaining spastic hemiparesis on the left side.

On November 7, ten weeks later, he returned to the hospital for the third time in a comatose condition with generalized convulsions, a temperature of 104 F. and a pulse rate of 140. For six weeks following his last discharge there had been steady improvement, gain in weight, headache only occasionally and no vomiting or convulsions. For about three weeks the condition had remained unchanged, but one week before his present entry the patient had begun to lose ground. Headache had returned with increasing severity, he had lost considerable weight and had felt much weaker. Some change in personality was also noted. A hypodermic injection of morphine given in the hope of relieving the headache was followed by loss of consciousness, and the patient was hurriedly brought back to the hospital.

Shortly after admission he had eight generalized convulsions. Examination revealed the following symptoms: The neck was stiff, the area of decompression tense, the right pupil larger than the left and the right eyeball more prominent. Both eyes were deviated to the right, and the fundi showed blurring of the disks. The patient was incontinent, and the hemiplegia on the left was unmistakable. There was still some purulent discharge from the incision over the right frontal

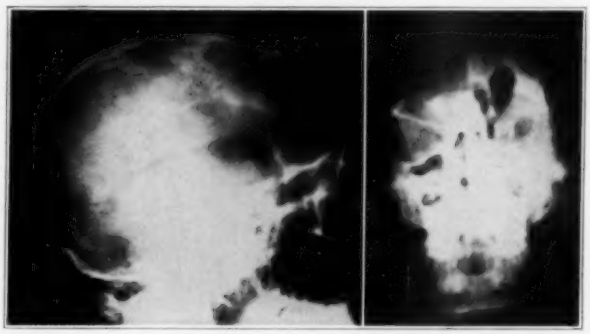

Fig. 3 (case 3).—Ventriculograms showing displacement of the ventricles to the left. Note also the flattening and depression of the right ventricle.

sinus. The white cell count showed 17,300 with 88 per cent polymorphonuclears. The spinal fluid was turbid; it contained 305 cells per cubic millimeter, all polymorphonuclears. The sugar content was 78 mg. per hundred cubic centimeters. No organisms were found on smear or culture.

The following day the patient was less confused mentally though at times he gave evidence of hallucinations. The neck was not as stiff; the temperature had dropped to 99.8 F. and the pulse rate to 90. The patient was no longer incontinent, and surprisingly, he began to move the left arm and leg. Within the following ten days the condition improved rapidly; the spinal fluid became clear, and the cell count showed only 5 lymphocytes.

Because of the purulent discharge from the frontal sinus and the roentgen evidence of osteomyelitis of the right frontal bone it was decided to approach the problem in two stages. In the first the purpose was to get rid of the osteomyelitis. Using the already existing incisions of the frontal operation and the subtemporal decompression on the right side a craniotomy was performed to include all the diseased part of the right frontal bone. The exposed dura showed one small area of granulation about the motor arm center. The diseased bone was discarded (fig. 4), the discharging sinus region cleaned up and the skin sewed back in a double layer immediately over the dura.

Progress for the next two weeks was satisfactory; the wound healed by primary union, and the spinal fluid remained normal. It then seemed safe to perform the second stage of the operation, namely, drainage of the cerebral abscess. The operation consisted of an incision over the motor area for the right arm. At a depth of 4 cm. an exploratory needle struck pus. The exposed dura was sealed

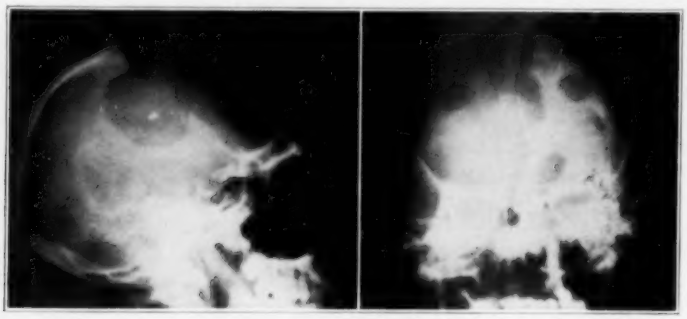

Fig. 4 (case 3).—Roentgenogram, showing the extent of bone removal necessary to eradicate the osteomyelitis.

Fig. 5 (case 3).—The Mosher drain in the frontal abscess.

off with an electric coagulating knife, and a cone of dura, cerebral tissue and abscess wall was cored out; the Mosher drain was then inserted into the cavity of the abscess (fig. 5). Culture from the abscess showed a growth of Staph. albus-haemolyticus.

There was a moderate discharge of pus from the basket, and within a fortnight the basket began to pulsate and the general status seemed much improved. A week later the patient was allowed to be out of bed; he walked with a hemiplegic

gait but was comfortable in other respects. A month after its insertion the drain was removed and healthy granulations which pulsated freely came to the surface, and there was no tendency for the brain to herniate. On December 9, when the patient was discharged, he was free from headache and the fundi were normal, but some facial weakness, a spastic paresis, overactive deep reflexes and a Babinski sign were present on the left side.

For almost two months he seemed fairly well but on Feb. 8, 1934, he was readmitted because of a return of headache, which had become increasingly severe during the preceding week. There were no spells of vomiting or convulsions. The findings at this time were in no way different from those at his previous discharge. The wound was not tense; there was no fever or bradycardia, and the fundi were normal. I was at a loss to account for the headache. The spinal fluid showed a pressure of 200 mm. but was otherwise normal. The removal of 15 cc. and 50 cc. on two separate occasions caused the wound to sink in for a brief interval but did not influence the headache. Intravenous administration of dextrose failed to bring relief. In an attempt to gain additional information encephalograms were made; unfortunately they showed little air in either ventricle, but the roentgenograms showed small coalescing islands of newly formed bone over the right frontal defect.

The headache continued, unassociated with fever, vomiting or bradycardia. It was sufficiently severe to cause the patient to display suicidal tendencies. As a desperate measure a needle was introduced through the scar of the previously drained area, and to the astonishment of the attending physicians 120 cc. of thick, foul pus was aspirated which on culture again showed Staph. albus-haemolyticus. There was only slight relief of the headache. On the following day 20 cc. of pus was removed from the same region, and on the following day 25 cc. more was evacuated with only moderate symptomatic improvement.

On March 1, operation was performed with the patient under anesthesia with avertin and procaine hydrochloride. The old incision was reexplored, and at a distance of 4.5 cm. from the surface a thick, shaggy, reddish, fibrous membrane—evidently the capsule of the abscess—came into view. After four traction sutures had been placed in the capsule it was brought to the surface; the abscess cavity was entered, the pus was sucked out and the cavity was packed with 5 yards (4.57 meters) of 1 inch (2.54 cm.) gauze. The edges of the abscess wall were then sutured to the surface in the fashion of marsupialization of a cyst. On the following day the patient was free from headache. The packing was changed several times within the following week, and with each dressing the cavity of the abscess became smaller and smaller, but a cerebral hernia appeared along the medial side of the presenting abscess wall. Though the granulations of the abscess cavity were healing nicely, the cerebral hernia (fig. 6) increased in size, and with it came the return of the distressing headache.

Within the following two weeks matters went from bad to worse, and as intravenous administration of dextrose and repeated spinal drainage failed to have any convincing effect on the cerebral hernia, a fine needle was inserted in the right temporal region. This intervention was again rewarded by the evacuation of 90 cc. of thick pus and a prompt decrease in the size of the hernia. Five days later the hernia regained its previous size, and again aspiration was carried out with the removal of 95 cc. of pus, but this time 75 cc. of air was introduced into the abscess cavity. Roentgenograms (fig. 7) made shortly afterward showed that there was a single abscess cavity. They also showed its size and location and, what was most important, revealed that it was part of the cerebral hernia.

Several days later the hernia was incised, exposing an abscess cavity about the size of an orange; anesthesia with procaine hydrochloride was used. The cavity presented a red, granular, corrugated surface covered in part by thick

Fig. 6 (case 3).—Cerebral hernia.

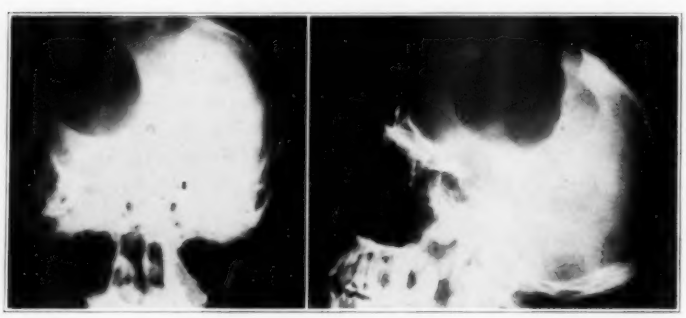

Fig. 7 (case 3).—Roentgenogram of the abscess cavity after injection of air (compare with fig. 6).

yellow pus. About 100 cc. of pus was aspirated, and the cavity was packed with gauze and four irrigation tubes. The headache promptly disappeared. Irrigation with surgical solution of chlorinated soda was kept up for over a week. The tissue from the cerebral hernia, which was really part of the abscess wall, showed a

chronic, suppurative, inflammatory process, the borders consisting of a necrotic mass of tissue infiltrated with polymorphonuclear cells and nuclear remnants. Much of this membrane showed beginning hyalinization.

During the next three weeks the granulations came closer and closer to the surface; the patient remained afebrile and free from headache, and on May 5 he was discharged.

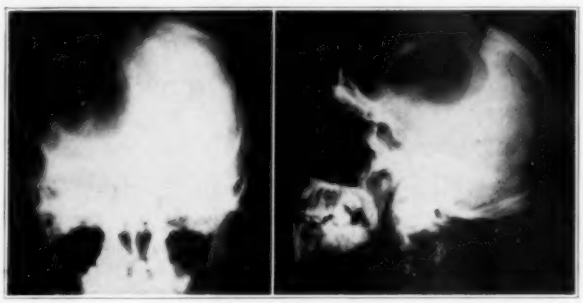

Fig. 8 (case 3).—Roentgenogram of the cavity of the recurrent abscess outlined by injection of air. Note the islands of osseous regeneration.

Fig. 9 (case 3).—Limits of healed incision and granulations of the abscess wall. Note the absence of deformity in spite of the extensive removal of bone.

He was readmitted on August 22 because of recurrent sore throat. After leaving the hospital in May he had been free from headache and his general condition had improved, but his progress was somewhat impeded by a chronic inflammation and infection of the tonsils. The tonsils, anesthetized with procaine hydrochloride, were removed, and for a week he had a stormy course with fever and generalized pains. At the end of that period he again complained of headache and the wound appeared tense, but the temperature was normal. The leukocyte count and sedimentation time were both normal. Aspiration of the abscess cavity brought forth

120 cc. of pus. At this time 50 cc. of air was introduced, and roentgenograms of the skull showed the location of the recurrent abscess (fig. 8). The following day 60 cc. of pus was withdrawn with only moderate relief of the headache. The abscess cavity was reopened; the residual 100 to 125 cc. of purulent material was sucked out; irrigation tubes and packing were inserted into the cavity, which presented a uniformly healthy granulating surface, and daily irrigations were instituted. Culture of the abscess material showed Staph. albus-haemolyticus. Progress was satisfactory, and healing was prompt as the granulations approached the surface of the wound (fig. 9). Since October 9, when he was last discharged, the patient has gained 20 pounds (9.1 Kg.); he is free from headache and is able to get about unassisted.

Comment.—The unusual situation of the abscess of the frontal lobe in this case is best explained as secondary to an osteomyelitis of the frontal bone which in turn was brought about by a suppurative frontal sinusitis.

Fig. 10 (Dr. Dandy's case).—Roentgenogram showing the proximity of the cerebral abscess to the lateral ventricle.

The injection of air into the cavity of the cerebral abscess was a desperate measure to clarify the problem. It was realized, however, that the abscess wall was by this time quite thick and that the tension of the remaining purulent material plus the air injected was much less than the preexisting tension.

After a careful and fruitless search for reports of a similar procedure, I communicated with Dr. Walter Dandy, who replied: "Only once have I injected air into an abscess cavity, and that was in the early days when I was enthusiastic about it." He also sent me the roentgenogram (fig. 10) and gave me permission to use it.

The exact visualization of the abscess cavity, its size, shape and location and the knowledge that there was only a single abscess led to the satisfactory drainage of this obscure suppurative process.

CASE 4.—*Trauma of the head; suspected meningitis or pneumonia. Headache, papilledema, left hemiparesis and left homonymous hemianopia. Possibility of cerebral abscess and of neoplasm considered. Abscess of the temporal lobe revealed through aspiration. Use of the Mosher drain. Recovery with some secondary optic atrophy and left homonymous hemianopia.*

On April 27, 1934, a schoolboy, aged 15, was referred to the Brooklyn Jewish Hospital by Dr. J. L. Abramson because of headache, drowsiness and weakness on the left side.

The patient was perfectly well until March 11, when he fell down some stairs and struck his head against the stoop. This was followed by dizziness for some time, but there was no loss of consciousness. Two days later the temperature rose to 105 F.; there was some stiffness of the neck, and vomiting occurred twice. The patient was admitted to a neighboring hospital with the provisional diagnosis of pneumonia or meningitis. Although there were some signs suggestive of pulmonary infection, roentgenograms of the chest showed normal conditions. The patient soon began to complain of pain in the suboccipital region, of parietal headache and of pain across the lower part of the back. Two weeks after the onset of the illness he spoke of seeing double; vision became blurred, and he noted that he had difficulty in seeing objects to the left. Any attempt at walking showed a tendency to fall to the left. After three weeks the patient became so irritable, noisy and unmanageable that it was thought advisable to take him home. While at home he had seizures which started with a scream and were followed by convulsive tonic and clonic movements of both hands and feet. He had as many as three attacks a day; none lasted more than five minutes and they were never associated with loss of consciousness. At about the same time he complained of pain in the left ear, but examination disclosed no abnormality. For two weeks he was incontinent of urine and feces, and at times he complained of severe abdominal cramps. Since the onset of the illness he lost 27 pounds (12.2 Kg.).

Examination showed a frail, irritable boy who lay limp and moaned most of the time. There was marked wasting. He appeared drowsy and complained of distressing headache. Vomiting was frequent and forceful. The neck was rigid. The pupils were dilated, the right more so than the left, and there was horizontal nystagmus to the right with slight limitation in upward gaze. The fundi showed bilateral papilledema of 3 diopters with dilated tortuous vessels, hemorrhages and exudate. Gross tests disclosed an unquestionable left homonymous hemianopia. There was no discharge from either ear, and the hearing was good. The uvula deviated to the left. In spite of the pronounced weakness of the left arm and leg, the deep reflexes were equally diminished, and there was no Babinski, Gordon or Oppenheim sign. Astereognostic sense in the left hand was definitely impaired. The Kernig sign was present on both sides. The abdominal responses were sluggish bilaterally.

The temperature ranged from 97 to 101 F., the pulse rate from 90 to 60 and the respiratory rate from 18 to 20. The roentgenograms of the chest showed that both pulmonary fields were clear, and those of the skull and cervical region of the spine indicated normal conditions. The blood pressure was 120 systolic and 75 diastolic. The white blood cells numbered 11,250 with 88 per cent polymorphonuclears. There were 3,700,000 red cells, and the hemoglobin was 68 per cent. The sugar content of the blood was 94 mg. per hundred cubic centimeters; the urea nitrogen, 11.4 mg., and the carbon dioxide, 50.7 volumes per cent. The Wassermann and Kahn reactions of the blood were negative.

While he was in the hospital the patient had a seizure which started with a shriek and was accompanied by loss of consciousness. The left pupil became

dilated, and there were clonic movements of the left arm and leg. Lumbar puncture yielded clear fluid, under a pressure of 35 mm., with 7 lymphocytes per cubic millimeter. The sugar content was 71 mg. per hundred cubic centimeters. The Wassermann reaction was negative. No organisms were found on smear or

Fig. 11 (case 4).—Cerebral hernia after the Mosher drain was extruded.

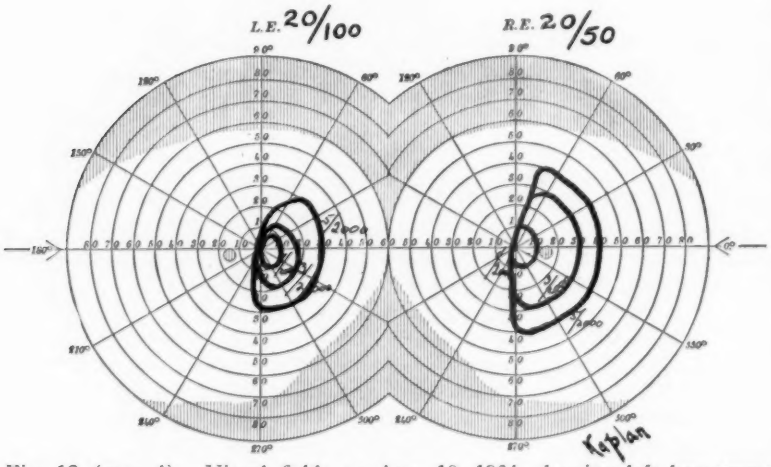

Fig. 12 (case 4).—Visual fields on Aug. 10, 1934, showing left homonymous hemianopsia with sparing of the macula.

culture. Two diagnoses were considered at that time: intracranial neoplasm and cerebral abscess.

On May 4 a large temporoparietal flap was outlined on the right side with the plan of first exploring for an abscess of the brain in the posterior part of the right temporal lobe and, if no abscess was found, of proceeding with the exploration for a neoplasm. The first trephination was therefore made over the right temporal region in the posterior limb of the incision, and thick yellow pus was

obtained at a depth of 4 cm. The stylet was immediately replaced to prevent the collapse of the abscess; the bony defect was enlarged to the size of a half-dollar; the dura was coagulated at the margin of the bone, and then a core of dura, brain and abscess wall was removed en bloc with an electric coagulating knife, and the Mosher drain was inserted into the depth of the cavity. Culture of the purulent material obtained showed streptococci.

On the following day the patient had a severe spell of vomiting, but in the main he seemed improved. Mentally he was more alert; the neck was not as stiff and the left arm and leg could be moved with greater ease. Within the following four days the temperature was normal, vision subjectively was improved, and both fundi showed evidence of recession. One week after the operation the drain was discharged spontaneously, and behind it was a nonpulsating cerebral hernia of moderate size. Power in the left arm and leg improved daily. Two weeks after the operation the hernia began to pulsate; the disks were almost flat, and the patient was able to sit up in bed. Five days later he was out of bed and was able to walk with some assistance. At the time of discharge on July 7, thirty-three days after the drainage of the abscess, there remained only a slight weakness of the left side of the body, some astereognosis in the left hand, secondary optic atrophy in both fundi and a small pulsating cerebral hernia (fig. 11) which was rapidly receding. The patient weighed 104 pounds (47.2 Kg.), showing a gain of 19 pounds (8.6 Kg.) since the operation.

At the time of writing, seven months after discharge from the hospital, the wound is completely healed, the boy is back at school, weighs 134 pounds (60.8 Kg.) and has grown in height. Except for some secondary optic atrophy and a remaining left homonymous hemianopia there are no residual abnormal neurologic signs (fig. 12).

Comment.—This case presented a difficult problem in the differential diagnosis between a cerebral abscess and an intracranial neoplasm. The history of fever and the sickly appearance favored the diagnosis of abscess, yet it was difficult to establish a primary focus for such an infection. The evidence pointing to pulmonary involvement or infection of the upper respiratory tract was not convincing. A rare instance of a mild injury of the head being followed by a cerebral abscess has also been considered as a possible explanation.

CASE 5.—*Otitis media without involvement of the mastoid. Headache, vomiting, papilledema and left homonymous hemianopia. Unsatisfactory drainage with the Mosher drain. Recurrence of abscess; pneumographic visualization; replacement of the Mosher drain. Recovery with residual defect of the visual field.*

On June 1, 1934, a schoolgirl, aged 12, was referred to the Mount Sinai Hospital by Dr. Keschner with a diagnosis of abscess of the right temporal lobe.

Three years before admission she had lobar pneumonia from which she made a complete recovery. In the beginning of April 1934 she had a mild "cold" with a slight nasal discharge. Four weeks later she complained of severe pain in the back of the neck, and at this time the posterior cervical glands on both sides were tender. The temperature rose to 101 F., and the patient began to complain of frontal headache and pain in both ears. There was only a slight sanguinopurulent discharge from the right ear which cleared up without any treatment.

Three weeks before admission after intranasal administration of ephedrine the patient had a convulsion and remained unconscious for five hours. During that

time she was perfectly quiet except for automatic wiping of the nose. A second convulsion immediatley followed return to consciousness, and the patient was taken to a neighboring hospital, where a lumbar puncture was performed. Five days later she was discharged apparently well. She remained so for three days, and then the frontal headache and pain in the back of the neck returned, becoming so intense that the patient was unable to sleep. Ten days before admission there was a spontaneous profuse discharge from the right ear, and soon there followed attacks of transient diplopia, vomiting, malaise and fever. On careful questioning the patient stated that at times she smelled something "funny" like tincture of benzoin.

The child appeared acutely ill; she was irritable and was given to crying spells. The left ear was normal; the right drum showed obscuring of the light reflex but no perforation or discharge. There was no tenderness over the mastoid. The right pupil was slightly smaller than the left and reacted sluggishly to light. There was slight but definite weakness of both external rectus muscles. The fundi showed bilateral papilledema, the condition being more advanced on the right, and the visual fields gave evidence of a homonymous defect in the inferior left quadrant. The following findings were present on the left side: facial weakness, mild paresis, overactivity of the deep reflexes, diminution of abdominal responses, a Babinski sign and slight diminution of pain, temperature and vibration sense though the astereognostic sense was intact.

Though the spinal fluid showed a pressure of 240 mm. and 150 cells per cubic millimeter, 84 per cent lymphocytes and 16 per cent polymorphonuclears, there was no stiffness of the neck and no Kernig sign.

On the fourth day after admission, after she had complained of headache all morning, the patient suddenly went into coma. Lumbar puncture at that time showed a pressure of 260 mm. which after the withdrawal of 8 cc. of fluid dropped to 150 mm.; this was followed by a prompt return of consciousness. It seemed evident that it was not safe to procrastinate any longer, and therefore under local anesthesia aspiration of the right temporal lobe was carried out. The abscess was located more anteriorly than had been anticipated, and at a depth of 4 cm. there welled up thin yellow pus. The Mosher drain was inserted with some difficulty, and soon pus began to escape from the depths of the abscess cavity. Culture of the pus showed Streptococcus haemolyticus B, Staph. albus, diphtheroid bacilli and some anaerobic organisms. From time to time there was an escape of pus through the drain, but there was no convincing clinical improvement. On the fourth postoperative day the spinal fluid was cloudy, with a pressure of 320 mm. Three days later the general condition became worse; the patient complained of suboccipital headache and became drowsy; there were signs of meningeal irritation, and the cerebral hernia increased in size. The Mosher drain was removed and replaced by a rubber tube. Lumbar puncture on the following day showed a pressure of 360 mm., and after the withdrawal of 20 cc. the pressure fell to 150 mm. During the following two weeks there was a notable increase in the hemiparesis, an advance in papilledema on both sides and a marked left hemihypesthesia with a definite loss of vibration and position sense. The patient complained of pain in the right eyeball, and she vomited several times. Examination revealed in addition a complete left homonymous hemianopia and a bilateral palsy of the external rectus muscle.

Introduction of a probe at the upper portion of the cerebral hernia was followed by free escape of pus. A small drainage tube was inserted at this point for a distance of 5 cm., and a moderate amount of pus escaped. A week later the tube was expelled. In the meantime the headache, vomiting, pain in the eyeball

and fever subsided. The left arm could be moved more freely; the facial weakness was less marked; the fundi began to recede, and the general condition was on the mend.

On July 11 the patient was allowed to be out of bed; she walked with some assistance and favored the left side; the wound was granulating nicely and was almost flat, but no definite pulsations could be seen. The progress was satisfactory for the following ten days; then the "funny" smell like that of tincture of benzoin returned, and two days later there was a repetition of the olfactory hallucination. The temperature rose to 103.2 F. The patient had a chill lasting ten minutes which was followed by increasing headache and vomiting. The spinal fluid was cloudy, was under a pressure of 220 mm. and contained 2,000 cells per cubic millimeter with 90 per cent polymorphonuclears. For two weeks the febrile course persisted; the paresis and facial weakness which had been present on the left side became worse. The distressing headache and the pain behind both eyes which now radiated down the right side of the nose were all beyond control. Vomiting continued. The patient seemed more irritable and drowsy, and she

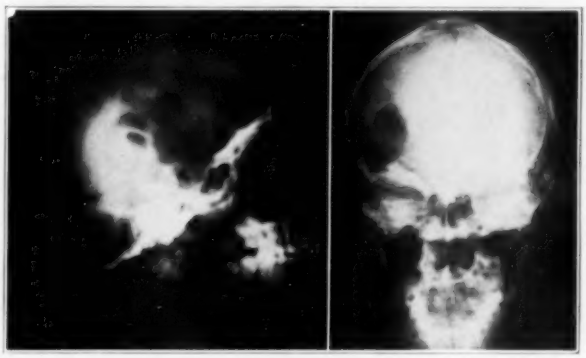

Fig. 13 (case 5).—Roentgenogram after injection of air. The air in the abscess cavity reveals its exact size, shape and location.

cried frequently. The disks showed that the intracranial pressure was steadily mounting. There was no stiffness of the neck and no Kernig sign.

On August 10 because of the desperate condition the wound was explored with a needle; at a depth of 4 cm. 30 cc. of foul, thick, purulent material was aspirated, and during this procedure the patient complained of pain in the teeth. Pulsation of the wound was clearly visible after the withdrawal of the pus, and the condition seemed much improved. Three days later a similar procedure was carried out, and after the withdrawal of 30 cc. of pus, 20 cc. of air was injected. Roentgenograms of the skull (fig. 13) clearly showed the size, position and shape of the abscess cavity. In the morning of the same day the abscess was opened widely, and a specially long Mosher drain was inserted. The abscess cavity contained about 100 cc. of pus.

On the next day the patient was given a transfusion of 250 cc. of citrated blood. There was a moderate discharge of foul-smelling pus through the drain; this diminished in quantity, and soon healthy granulations replacing the shaggy membrane of the abscess wall began to fill the meshes of the drain (fig. 14). The improvement following the last insertion of the Mosher drain was gratifying. Fever, headache and vomiting disappeared within forty-eight hours, and during

the ensuing week there was sufficient return of motor power of the left arm and leg to enable the patient to feed herself and walk without assistance.

On September 24 injection of iodized poppy-seed oil 40 per cent through the drain failed to show any communication with another abscess (fig. 15). Two days later the drain was removed. For the next four days there was some headache, vomiting and slight fever. Gentle probing of the wound yielded some yellow, blood-tinged fluid on three separate occasions. From then on the patient improved steadily.

Fig. 14 (case 5).—The Mosher drain in the right temporal lobe (compare with fig. 15).

Fig. 15 (case 5).—Mosher drain in the abscess cavity. Iodized poppy-seed oil 40 per cent failed to show any communication with a secondary abscess.

She was discharged on October 11, at which time she was free from symptoms; the wound was clean and pulsated freely; the fundi were normal; the visual fields showed a left homonymous hemianopia, and the left leg still showed a slight weakness.

At the time of writing, four months after her discharge, the patient has gained 15 pounds (6.8 Kg.), has no complaints and is back at school doing well in her studies. Except for some optic pallor and a left homonymous defect of the visual field the neurologic status is normal.

Comment.—In this case a temporosphenoid abscess secondary to inflammation of the middle ear without involvement of the mastoid was rightly suspected. The first attempt at drainage was not entirely satisfactory, probably because of faulty placement of the Mosher drain.

After pneumographic visualization of the abscess, operation presented no difficulties, and the drain was so placed that good drainage followed and recovery was prompt.

SUMMARY

A report of five cases of cerebral abscess in which recovery followed a fairly consistent regimen is presented. Although the series is too small to permit definite generalizations, the factors which have contributed to the successful outcome in these patients have been enumerated.

These cases also illustrate the fact that complications may arise after the initial localization and drainage of a cerebral abscess and that constant personal attention, which will determine when a revision of method may be desirable, is of paramount importance to the successful outcome of the individual case.

Three of the patients had an abscess of the temporal lobe complicating otitis media; one had an abscess of the frontal lobe secondary to osteomyelitis of the skull and suppurative frontal sinusitis. The abscess of the temporal lobe in the fifth patient was in all likelihood of metastatic origin.

Four of the patients have returned to their previous calling and are free from symptoms; one has been left with hemiparesis but is able to get about unassisted.

Dr. Ira Cohen granted permission to report case 5 from his service.

ACUTE CEREBELLAR ABSCESS

REPORT OF A CASE

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The unusual opportunity recently occurred to observe the complete course of a cerebellar abscess in a hospitalized patient from the onset of otitis media, which caused the abscess, to recovery following drainage. The clinical history of this case is reported because it is highly instructive in its bearing on the treatment of the condition.

According to Eagleton,[1] the average mortality from abscess of the brain is still high, 75 per cent or more. The statistics of Coleman,[2] however, who obtained nineteen recoveries in twenty-eight cases (a mortality of only 31 per cent), suggests that the average number of deaths is unnecessarily high. Faunce and Shambaugh[3] more recently reported six consecutive cases with only one death.

The usual high mortality from abscess of the brain is in part due to failure to make a timely diagnosis. Eagleton[1] emphasized that the literature is filled with reports of cases in which a diagnosis of abscess of the brain was not made "because of the absence of symptoms of brain abscess," whereas a careful review of the cases would show that in reality the symptoms were present and that if they had been properly interpreted the correct diagnosis could have been made.

The possibility of the presence of an abscess of the brain should always be thought of when cerebral symptoms develop during or shortly after otitis media, paranasal sinusitis, abscess of the lung or bronchiectasis. The initial symptoms of cerebral suppuration are usually mild, consisting of slight fever, headache and vomiting. Localizing neurologic changes appear only as the abscess becomes well developed, and the symptoms and signs of cerebral compression may not appear until shortly before death. While delay in operative intervention is advantageous in the early stages of abscess of the brain, delay of even a day in the later stages may be fatal.

From the Municipal Contagious Disease Hospital.

1. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922.

2. Coleman, C. C.: Brain Abscess: Review of Twenty-Eight Cases with Comment on Ophthalmologic Observations, J. A. M. A. **95**:568 (Aug. 23) 1930.

3. Faunce, C. B., and Shambaugh, G. E., Jr.: Abscess of the Brain Following Mild Transitory Otitis Media, Arch. Otolaryng. **17**:673 (May) 1933.

The high average mortality can be ascribed in part to the treatment. Coleman believed that the tendency to do too much in operating is responsible for many deaths. He pointed out that in the majority of cases the patient with an abscess of the brain who dies postoperatively dies not as a result of the abscess itself but as a result of acute, traumatic encephalitis in the surrounding tissue. Dandy [4] stated that the mechanism of death in most cases is undoubtedly increased intracranial pressure and that in many instances the patient dies as a result of the treatment rather than because of the abscess itself. Thus, for cerebellar abscess, because of the more limited space, the mortality is about 90 per cent as compared with the mortality from cerebral abscess, which is about 70 per cent. Dandy said that he relies on simple evacuation of an abscess through a needle, sometimes a single tapping sufficing to cure a chronic abscess of long standing, although sometimes repeated tappings are necessary. Coleman reported that he supplements simple tapping with drainage through a rubber catheter which is introduced into the abscess cavity. By either method trauma to the intact tissue of the brain is reduced to a minimum and the usual postoperative increase in intracranial pressure with massive herniation of the brain, which occurs so often with a bolder surgical attack, is avoided.

Abscesses of the brain are often spoken of as acute or chronic, acute referring to the stage of initial cerebral invasion and chronic, to the stage of encapsulation. Cerebral invasion may be embolic (if from an abscess of the lung or bronchiectasis), by direct contiguity (usually from chronic otitis media) or by a retrograde thrombophlebitis of the pial veins, which extend from the region of the middle ear and the mastoid through the dura (the usual pathway of infection in cases of abscess of the brain following acute otitis media). This retrograde thrombophlebitis may lead to acute suppurative meningitis, it may remain localized in the veins and eventually heal, or at some point in the white substance of the brain where the blood supply is poorer than in the cortex, it may lead to local necrosis of brain tissue, possibly by the formation of an infarct. Acute inflammation of the surrounding brain tissue ensues, while an abscess forms in the small area of necrotic tissue. After several weeks a capsule begins to form around the abscess, and the chronic stage begins. Operative intervention during the acute stage only interferes with nature's attempt at encapsulation; so drainage should be delayed until the chronic stage has been reached and encapsulation has occurred.

The following case report concerns a child who was admitted to the hospital during the eruptive stage of scarlet fever. During hos-

4. Dandy, W. E.: Treatment of Chronic Abscess of the Brain by Tapping, J. A. M. A. **87**:1477 (Oct. 30) 1926.

pitalization acute suppurative otitis media developed, complicated by thrombosis of the lateral sinus and followed by the development of a cerebellar abscess. By reviewing the clinical course in this case one can reconstruct and visualize the pathologic changes which occurred at each stage from the initial cerebral invasion to encapsulation, drainage and recovery.

REPORT OF A CASE

R. M., a 6 year old boy, was admitted to the Municipal Contagious Disease Hospital on Nov. 13, 1933, the sixth day after the onset of scarlet fever, with a temperature of 103.6 F. (rectal). Examination revealed a heavy punctate rash over the trunk, neck and limbs; a strawberry tongue; red and swollen tonsils, uvula and pharynx covered with a yellowish-white membrane; a purulent nasal discharge, and cervical adenitis. The white blood cell count was 32,800. The child was critically ill, but with no specific treatment the temperature slowly subsided and the condition gradually improved until the twelfth day after admission, November 25, when he complained of pain in the left ear and the temperature rose to 103.8 F. The drum membrane ruptured spontaneously twelve hours later with a profuse discharge, and the temperature became normal.

Except for desquamation and persistent discharge from the left ear, the child's progress was uneventful until the nineteenth day of the otitis media, when the temperature suddenly rose to 105 F., followed by a fall to 102.2, a rise to 105.8, another fall the next day to 100.4 and then a sharp rise to 106. A blood culture was positive for Streptococcus haemolyticus. There was slight tenderness along the course of the left internal jugular vein. There were no symptoms except occasional vomiting and listlessness, and no other cause for the fever could be found. A diagnosis of thrombosis of the left sigmoid sinus was made and prompt operation advised.

On December 15 the left internal jugular vein was exposed and found to be collapsed above the facial vein and surrounded by a chain of inflamed lymph nodes. It was ligated above the facial vein. The mastoid cells were found to contain granulations and a little pus, and there were signs of beginning decalcification of the cell partitions. The sinus plate, however, was intact, and the wall of the sigmoid sinus was grayish and thickened but free from granulations. Incision of the sinus revealed a thrombus, which was removed downward as far as possible with no bleeding and backward to the beginning of the lateral sinus, where bleeding occurred freely.

Since the sinus plate was intact and there was no perisinuous abscess, it was surmised that the pathway of infection to the lateral sinus was hematogenous by means of thrombophlebitis of the small venules from the mastoid which penetrate the sinus plate and enter the sinus.

The operation, supplemented by a transfusion of 250 cc. of whole blood the next day, was followed by a marked improvement in the child's general condition. His appetite was good, there was no more vomiting and he looked and felt better, though for ten days there continued to be a low grade fever with the temperature between 102 and 103 F. every afternoon. Following a second blood transfusion the temperature subsided to normal and for several days he appeared to be well on the road to recovery.

On December 31, seven weeks after admission (five weeks after the onset of otitis media and sixteen days after operation on the sigmoid sinus), the child's temperature rose to 103 F. and he vomited. For a week the temperature continued

as high as 103.4 F., but there were no other symptoms and no cause for the fever could be ascertained. A blood culture was negative at that time.

On Jan. 6, 1934, he complained for the first time of headache and again vomited. Neurologic examination revealed nothing abnormal except a poorly sustained ankle clonus, which was more marked on the left side. The temperature that day subsided to 100.2 F. and the following day was normal, but the child again vomited, appeared listless and tired and complained of headache. Neurologic examination (January 7) gave negative results except for definite, slight adiadokokinesis of the left hand. The eyegrounds were normal and the mastoid wound was healthy and granulating well. Another blood transfusion was given but the child continued for the next few days to be listless and appeared pale. The temperature remained normal, but the pulse rate, which had been between 110 and 130 since admission, dropped at times to as low as 80.

On January 14 the child was worse, with increased vomiting and pain in the left eye, and more listless. A sustained ankle clonus was present on the left side, both pupils were widely dilated and failed to react to light, and the adiadokokinesis of the left hand was more marked. On January 15 there was spontaneous nystagmus to the left and the child seemed weaker and more lethargic. A lumbar puncture was performed carefully and because of the possibility of a cerebellar abscess, only a few cubic centimeters of fluid under moderately increased pressure was withdrawn. There were 42 cells, all lymphocytes; the reaction to the Pandy test for globulin was strongly positive, the sugar content was normal and a culture gave negative results.

On January 16 the child was apathetic but mentally clear. He had become emaciated in only a few days, the rapidity with which emaciation had developed being out of proportion to the loss of nourishment from vomiting. There was spontaneous nystagmus to the left, intermittent and irregular, with slight rotary and slight but definite vertical nystagmus at times. The eyes at rest tended to remain deviated to the right. Adiadokokinesis and ataxia of the left hand were more marked. For the first time the vomiting was projectile, and the pulse rate was as low as 66 at one time in the afternoon. The eyegrounds were normal at 8 o'clock in the morning, but in the afternoon, for the first time, they showed definite blurring of the margins of both disks, the veins were tortuous and engorged and a small hemorrhage appeared on the temporal margin of the left disk.

Because of these signs of rapidly increasing intracranial pressure, immediate decompression of the left cerebellum was carried out under local anesthesia induced by procaine hydrochloride, supplemented by light ether anesthesia. The diagnosis lay between abscess of the brain and encephalitis adjacent to the thrombosed lateral sinus.

While the periosteum was being retracted at the beginning of the operation, respiration ceased. Epinephrine and caffeine injected subcutaneously and artificial respiration for about one minute were necessary before breathing was resumed. An area of the cerebellar dura 5 cm. in diameter was exposed posterior to the sigmoid sinus. The dura did not seem to be under greatly increased tension, and the sigmoid sinus was soft to palpation at the knee, suggesting the presence of pus beneath it. When the wall of the sinus was incised, the scalpel entered abnormally soft, edematous brain tissue which bled readily. This was probed a short distance, but no sense of resistance was encountered. Iodoform gauze was placed against the dura, and the child was returned to bed.

Gradual improvement was immediately evident following decompression. There was no further vomiting or headache, the rapid development of emaciation abruptly ceased, the pulse rate became more rapid, and the child was considerably brighter

and stronger. The neurologic findings, however, continued to increase gradually: intermittent, irregular nystagmus, at times vertical; marked ataxia and adiadokokinesis of the left arm and leg; a tendency to lie on the right side with the knees drawn up, and deviation of the eyes downward, to the right or to the left. The changes in the eyegrounds gradually increased, with hyperemia and beginning elevation of the disks, blurred disk margins, patches of retinal exudate and dilated, tortuous veins with small extravasations of blood along them.

On looking back it was evident that the initial cerebral invasion began on Dec. 31, 1933 (fever and vomiting), the pathway of infection probably being the retrograde thrombophlebitis of the venules entering the lateral sinus from the cerebellum, similar to the pathway of infection to the lateral sinus itself two and one half weeks earlier. The initial thrombophlebitis was accompanied by systemic symptoms (fever), but after a week they subsided (the temperature was normal on Jan. 7, 1934) and the process might have stopped there. However, at one point in the brain the thrombophlebitis had led to local necrosis of the tissue. Edema of the involved and surrounding tissue ensued, with increasing signs of intracranial pressure. On January 16 the rapid increase in intracranial pressure would have caused death from medullary compression had not decompression been performed. Meanwhile, the familiar process of the formation of an abscess was proceeding, resulting in progressive neurologic impairment.

A definite diagnosis of cerebellar abscess on the left was made on January 23. It was decided to delay exploration as long as improvement in the symptoms of increased intracranial pressure continued so as to allow walling off and encapsulation of the abscess to occur and the encephalitis to subside. Immediate exploration was advised if vomiting, a slow pulse rate, headache or coma supervened.

On January 24 the patient was found to be unconscious and rigid with slight twitching movements. The pulse rate was 64. Epinephrine was administered, and the child became relaxed and conscious in twenty minutes. An intravenous injection of 20 cc. of a 50 per cent solution of dextrose was then given. An hour and a half later (twenty-five days after the first evidence of intracranial invasion) the left side of the cerebellum was explored with a Cushing needle through a ½ inch (1.27 cm.) incision through the inner wall of the perpendicular portion of the sigmoid sinus. A definite sense of resistance was encountered 3.5 cm. from the surface at an angle of 30 degrees posteriorly, and when this resistance was perforated, 5 cc. of thin, green pus escaped. The patient, who had again become unconscious shortly before he was taken to the operating room so that no anesthesia was necessary, quickly brightened. A small (no. 14) rubber catheter was inserted 4 cm. along the path of the needle and sutured to the dura.

For a few days the child's condition improved, but there was practically no drainage from the tube. On January 29 the child again vomited and was listless and pale; in the afternoon he cried out and then became unconscious and made convulsive movements. Respiration was labored, and the pulse rate was 74. The drain was removed and the Cushing needle was reinserted. It entered an abscess cavity which was definitely larger, and 15 cc. of watery, cloudy fluid escaped under pressure, followed by thin pus. The catheter drain was reinserted. The child immediately regained consciousness and was improved.

On February 2 nausea and vomiting again occurred, accompanied by severe pain in the head. There was no drainage from the tube. The abscess was aspirated with the Cushing needle and the drain replaced. The child improved somewhat, but no pus came from the drain. On February 6 the child became very ill, moaned because of pain in the head, vomited and was weaker. In the afternoon he had a mild convulsion and the pulse rate fell to 68. The cerebellum was explored and pus was found 6 cm. from the surface, about 15 cc. being evacuated. An attempt to insert a catheter into the abscess cavity was unsuccessful. As it became apparent that the catheter must always have slid off to one side of the capsule into the softer, edematous brain tissue and had never penetrated the wall, it was decided to rely on repeated aspirations.

On February 8 the child had two convulsions and the abscess was aspirated. On February 11 he had a severe convulsion lasting for five minutes. He vomited, and to the neurologic signs were added paralysis of the right external rectus muscle and stiffness of the neck. The eyegrounds showed many large and small hemorrhages, the vessels were completely buried in exudate at the margins of the disks, there was bilateral papilledema of 4 diopters, and the child was so nearly blind that he could not recognize his parents.

It was evident that the patient's condition was gradually growing worse. Each successive aspiration was followed by less improvement although the total quantity of pus obtained was not increased. The abscess itself, therefore, was not becoming larger but the encephalitis of the surrounding tissue was increasing. The increased distance of the abscess from the surface (from 3.5 to 6 cm.) was proof of increasing encephalitis. The medulla and pons must have been pushed far beyond the midline by the greatly enlarged left cerebellar hemisphere for the abscess to have been so deep. This displacement of the pons resulted in the paralysis of the right external rectus muscle, the sixth nerve being caught against the apex of the right petrous. It was apparent that if constant, adequate drainage was not provided the child would soon die.

The brain was again explored with the Cushing needle on February 11. Extremely thick pus was encountered after a tough capsule had been penetrated 6 cm. from the surface of the left cerebellum. A grooved director was then inserted along the needle. The latter was withdrawn, and a long, thin-bladed scalpel was introduced along the director, slitting the tough capsule 6 cm. from the surface. A no. 18 rubber catheter was then inserted along the director into the abscess. For the first time the drain did not have a tendency to be forced from the cerebellum by the increased pressure but seemed to be firmly gripped by the capsule. The drain was sutured to the dura.

The next day, February 12, the patient was slightly improved, and for the first time pus was escaping along the outside of the drain. From this point on recovery was slow but uninterrupted.

On February 18 there was definite improvement in the ataxia and adiadokokinesis, the appetite was good, the child was alert and responsive and the stiffness of the neck was gone.

On February 25 the vision had improved so that he could count fingers at a distance of 3 feet (0.9 meter) with one eye and at a distance of 1 foot (0.3 meter) with the other. Considerable pus continued to escape from around the tube. The child was gaining weight.

On March 4 the ataxia and adiadokokinesis had all but disappeared, fingers were counted at 6 feet (1.8 meter), and the paralysis was nearly gone. The drain was beginning to protrude and ½ inch was cut off to permit it to be forced out as the encephalitis subsided and the abscess approached its original site. Purulent drainage from around the tube continued.

On April 15 the drainage had practically ceased, and all but 3 cm. of the tube had been forced out. The eyegrounds were normal, vision was good, and the child was up and about. Some spasticity of the left leg in walking was the only residual neurologic impairment.

By May 23 the tube had completely extruded itself, the wound had healed and the patient was discharged.

At the present writing, six months later, the child is normal in every respect

The remarkable recovery of function after so marked a neurologic impairment may be ascribed, in part, to the smallness of the incision ½ inch (1.27 cm.) made in the dura, thus preventing massive herniation of the cerebellum. Had a larger or a crucial incision been made, a large hernia would undoubtedly have developed, with further trauma to the delicate tissue of the brain, resulting in sloughing and permanent impairment of cerebellar function.

CONCLUSIONS

The stage of invasion of the abscess of the brain (January 1 to 7) was characterized by predominating systemic symptoms (fever) from the advancing thrombophlebitis, the symptoms of local cerebral involvement (vomiting) being minimal.

The second stage, that of early necrosis and encephalitis (January 7 to 16), was characterized by signs of rapidly increasing intracranial pressure (vomiting, headache, lethargy, slow pulse, choked disks, coma and convulsions), with localizing neurologic signs beginning to appear.

The third stage, that of encapsulation (January 17 to 24), was characterized by continued signs of increased intracranial pressure, but with the localizing neurologic signs predominating (irregular, spontaneous nystagmus, at times vertical; conjugate deviation of the eyes; adiadokokinesis and ataxia of the arm and leg on the same side; the assuming of the so-called cerebellar posture; weakness and partial atrophy of the muscles of the arm and leg on the same side, and rapid, general emaciation).

The stage of recovery (February 11 to May 23) was marked by rapid disappearance of the signs of increased intracranial pressure followed by a more gradual recovery from the neurologic changes.

Encapsulation was definitely developed three and one-half weeks after the initial cerebral invasion.

Repeated aspirations were not sufficient. Constant drainage was necessary.

The encephalitis surrounding the abscess was more threatening to life than the abscess itself. Had a larger drain been inserted or any attempt been made to suck out, inspect or pack the abscess cavity, the added encephalitis would probably have been fatal. In cases of acute abscess of the brain, drainage must be established with a minimum of trauma to the tissue. A rubber catheter makes a satisfactory drain, but it must actually penetrate the capsule.

A preliminary decompression will relieve the acute symptoms in the early stages and allow delay in exploring until encapsulation and walling off have occurred.

By limiting the dural opening to a simple ½ inch (1.27 cm.) incision, massive herniation is prevented.

The neurologic signs of abscess of the brain are due more to the associated encephalitis than to the actual necrosis of brain tissue, as evidenced by the complete restoration of cerebellar function in this patient.

122 South Michigan Avenue.

TRACHEAL DIVERTICULUM

OBSERVATIONS ON A CADAVER AND RESULTS OF HISTOLOGIC STUDY

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A diverticulum may occur in any hollow viscus, and much has been presented on this interesting abnormality relative to the esophagus, intestines and bladder. The reverse seems to be true with regard to diverticulum of the respiratory tract. Most observations were made and reported prior to the present century. This is rather interesting when it is recalled that much of the progress in the diagnosis and therapeutic procedures referable to the respiratory tract has been developed during this period. The Jacksons [1] in their complete presentation of benign tumors of the trachea and bronchi made no mention of diverticulum, though they considered retention cyst. Moersch [2] in a comprehensive review of the subject of fistula between the esophagus and tracheobronchial tree did not refer to diverticulum. Gerber [3] in a similar study described a pulsion type of esophageal diverticulum as having been present.

A gaseous tumor of the neck described in the literature as tracheal diverticulum, aerocele, bronchocele or tracheocele has long been known. Under the name of bronchocele or aerial goiter, Aetius in the fifth century mentioned tumors that formed in the neck during the strain of parturition. Plater [4] about the year 1600 gave a description of the condition which he clearly differentiated from goiter. Rokitansky [5] was one of the first to call the attention of the medical profession to this

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† Dr. Ziegelman died Oct. 12, 1934.

These observations were made during the midwinter clinical course in otolaryngology in Los Angeles, under the supervision of Dr. John F. Barnhill.

1. Jackson, Chevalier, and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1932, p. 43.

2. Moersch, Herman J.: Fistula Between the Esophagus and the Tracheobronchial Tree, M. Clin. North America **14**:715, 1930.

3. Gerber, Isaac: Esophago-Tracheal Fistula: Report of Case Probably Due to Syphilis, Complicated with a Pulsion Diverticulum, Am. J. Roentgenol. **6**:191, 1919.

4. Plater, cited by Coudray and Guisez: Tracheocele, M. Press & Circ. **157**: 344, 1918.

5. Rokitansky, Karl: Handbuch der pathologischen Anatomie, Vienna, Braumüller & Seidel, 1846, vol. 3, pp. 6, 7 and 11; Oesterr. med. Jahrb., 1838, vol. 16.

lesion. He believed that it developed as a result of chronic pulmonary and bronchial disease. Bland-Sutton [6] described it as occurring near the junction of the trachealis muscle with the cornua of the semirings of the trachea. Miller,[7] from a careful histologic study of the trachea, described the trachealis muscle as being transverse and composed of isolated bundles or bands. It was his belief and that of others [8] that between these isolated bands there was a relatively amuscular area which served as the site of election for the development of a diverticulum (fig. 1).

Most observers classify tracheal diverticulum as congenital and acquired, but others subdivide the acquired condition into the traction and the pulsion type, similar to the classification of the similar esophageal lesion.[9] Stibbe [10] in a recent article described the two types accordingly, stating that the congenital type has a narrow, and the acquired a wide-mouthed, orifice. Chiari [11] was inclined to believe that the congenital type is found at the location where supernumerary bronchi are prone to occur,[12] that is, where the fore and aft parts of the three parts of the trachea develop.[13] Though this was his conviction, he was unwilling from the observations he reported to state positively whether the condition was congenital or not. It was he who clarified the pathology and Mackenzie (while in Chiari's clinic) who presented the first case.[14]

Chiari's observations led him to believe that the majority of diverticula are acquired and are the result of an infection in the mucous glands of the posterior tracheal wall with the subsequent development of a diverticulum. He believed that the lesion may be single or multiple and that it may occur in any part of the trachea. He quoted Petit, who had studied a series of forty-one cases and who stated that a diver-

6. Bland-Sutton, John: Tumours, Innocent and Malignant, New York, Paul B. Hoeber, Inc., 1917, p. 716.

7. Miller, W. S.: The Trachealis Muscle, Anat. Rec. **7**:373, 1913.

8. von Czyhlarz, Ernst R.: Ueber ein Pulsionsdivertikel der Trachea mit Bemerkungen über das Verhalten der elastischen Fasern an normalen Tracheen und Bronchien, Centralbl. f. allg. Path. u. path. Anat. **8**:721, 1897.

9. Choruzenko, T. F.: Ein Fall von Tracheocele, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:537, 1928.

10. Stibbe, E. P.: True Congenital Diverticulum of the Trachea in a Subject Showing Also Right Aortic Arch, J. Anat. **64**:62, 1929.

11. Chiari, O.: Chirurgie des Kehlkopfes und der Luftröhre, Stuttgart, Ferdinand Enke, 1916, p. 217.

12. Aeby: Der Bronchialbaum der Säugethiere und des Menschen, Leipzig, Wilhelm Engelmann, 1880. Stibbe.[10] Chiari.[11]

13. D'Agutala: Mem. r. Acad. d. sc. d. Ist. di Bologna, 1885, vol. 6.

14. Kahler, Otto: Ueber Divertikel des Tracheo-Bronchialbaumes, Monatschr. f. Ohrenh. **45**:86, 1911.

ticulum may remain dormant between the trachea and esophagus until some added pressure such as a persistent cough or excessive force in the use of the voice causes it to extrude from between these structures. Others have stressed the importance of the use of musical instruments,

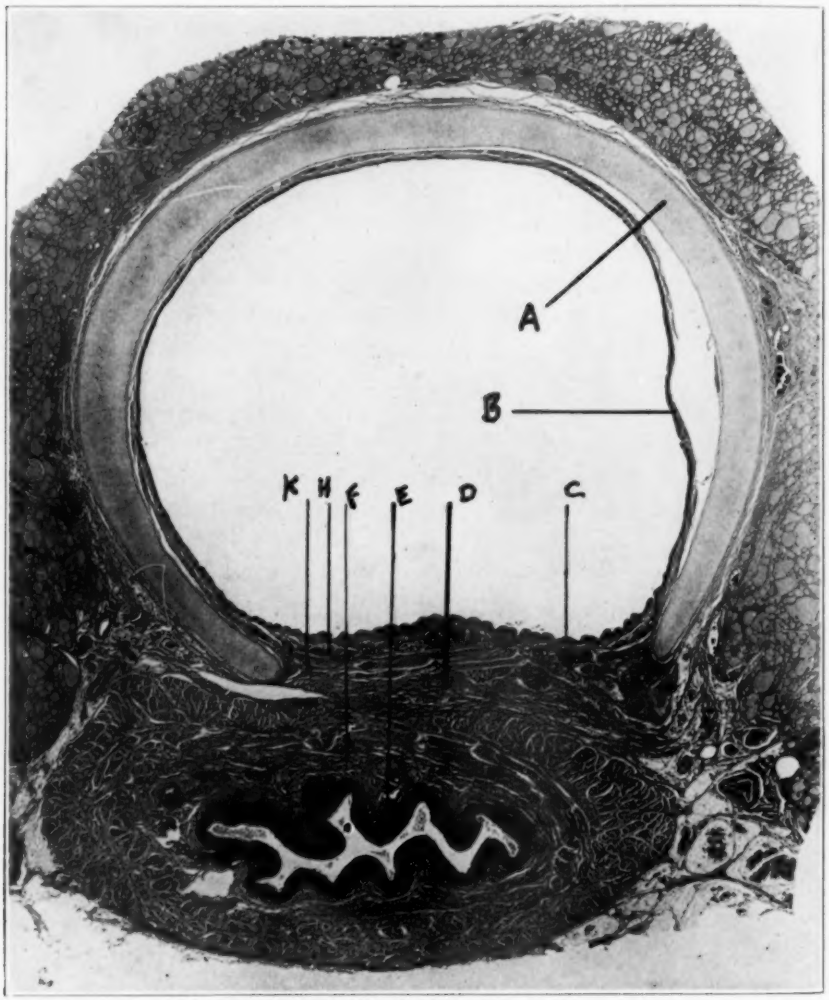

Fig. 1.—A photomicrograph of a section through the trachea and esophagus at the fifth tracheal ring. It depicts: *A*, cartilage; *B*, mucous membrane of the trachea; *C*, posttracheal glands; *D*, the trachealis muscle; *E*, mucous membrane of the esophagus; *F*, the body of the esophagus; *H*, posttracheal glands, and *K*, the right posterior cornua of the fifth tracheal ring and the location where the diverticulum described in the text developed. The section was taken from the respiratory tract of an infant and is magnified five times.

such as the large horns. Baron Larry [4] referred to cases that had come under his observation in minaret criers in Egypt and in drill sergeants. These men presented aerial tumors of the neck which were often of considerable size, consequent on hernia of the soft tissues in various segments of the trachea. To these lesions he gave the name aerial or vesicular goiter. Cases have been reported following terrific pressure, such as that caused by the bursting of a large shell. A soldier after

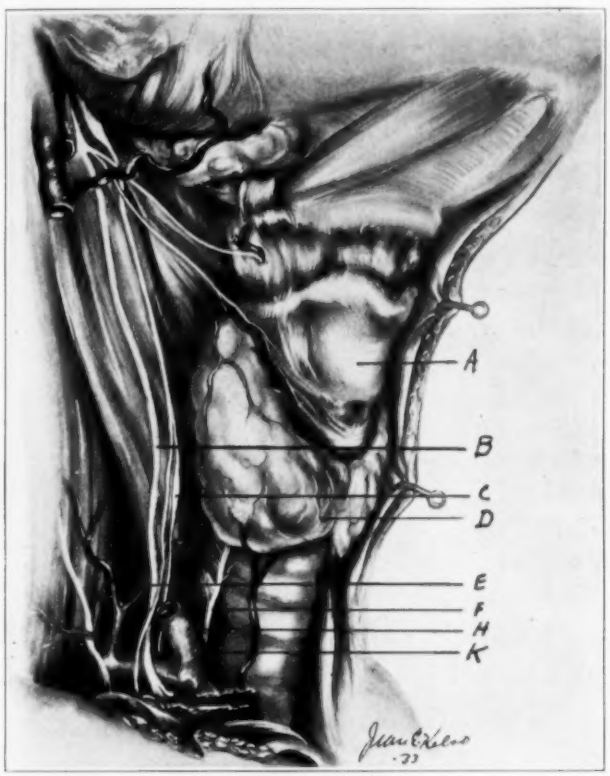

Fig. 2.—A drawing of a dissection to demonstrate structures of the lateral portion of the neck. *A* indicates the larynx; *B*, the right vagus nerve; *C*, the right sympathetic nerve; *D*, the right thyroid lobe; *E*, the right inferior thyroid artery; *F*, the right recurrent laryngeal nerve; *H*, the right common carotid artery severed, and *K*, the right tracheo-esophageal groove and the location of the diverticulum of the trachea at the fifth interspace.

being blown 40 feet (1219 cm.) was found to have a tracheal diverticulum. A clinical examination demonstrated that three rings of the trachea had been ruptured. A similar case was mentioned in which such a diverticulum developed following great effort on the part of a soldier to move a cannon. Laryngoscopic examination in these cases

Fig. 3.—A photograph of a specimen showing the opening into the diverticulum from the trachea.

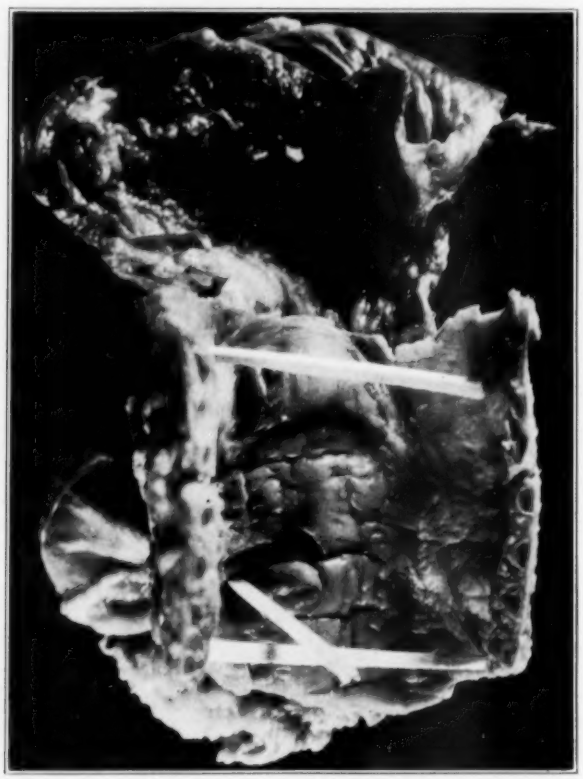

Fig. 4.—An enlarged photograph showing the opening into the diverticulum from the trachea at the fifth interspace. The trachea has been opened and its wall retracted.

revealed no abnormality except slight redness of the cords. No ulceration or opening in the trachea could be determined during the clinical examination.[4]

The following is a report of observations on a cadaver and histologic examination of a tracheal diverticulum:

During a routine dissection of the lower part of the neck a collapsed, irregular abnormal structure was found lying in the right tracheo-esophageal groove

Fig. 5.—A photograph showing the diverticulum, its comparative size and its location.

(fig. 2). It was somewhat adherent to the lateral esophageal wall and was dissected free with difficulty. On its anterior lateral wall was the right recurrent laryngeal nerve, which was adherent. The first conclusion was that an esophageal diverticulum was present, and enthusiasm was aroused owing to its position on the right side, which is a rather uncommon location. This pathologic condition was discussed with colleagues who were present during the course of the dissection (Dr. E. C. Sewall and others). All agreed that the orifice in the neck was too far anterior to be typical of an esophageal lesion. The trachea and esophagus were removed in toto, from the cricoid cartilage to below the episternal notch.

The esophagus was opened. No communication was found to exist with the sac. When the trachea was opened a communication which easily admitted a match was found (fig. 3). This was on the plane of the interspace between the fifth and the sixth tracheal ring in the posterior lateral tracheal wall near the right cornua of the sixth ring (fig. 4). The opening extended upward and communicated with the pouch. The sac was filled with water to prove the communication. When distended it measured approximately 6 by 4 by 3 cm. and held about 60 cc. of fluid (fig. 5).

Dr. Paul McKibben, head of the department of anatomy of the University of Southern California Medical School, and his associates, under whose supervision the microscopic sections were made, rendered the following report: "A number

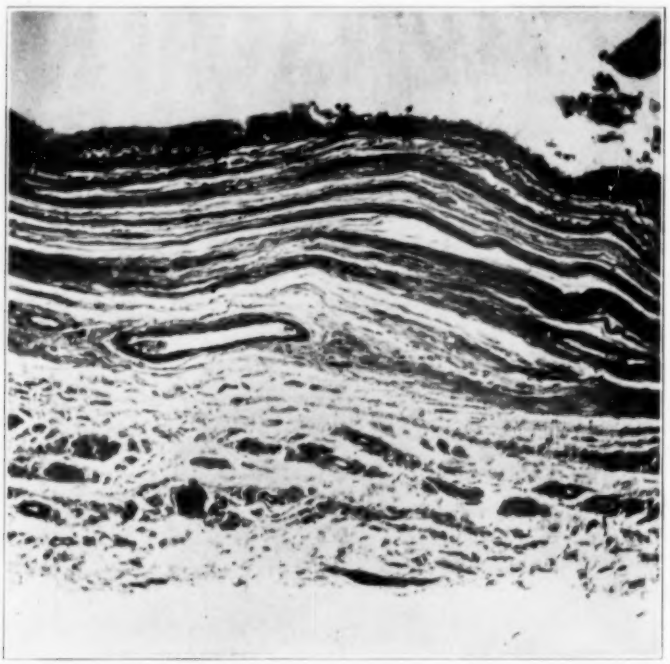

Fig. 6.—A photomicrograph (× 40) showing the histologic change through the wall of the diverticulum. Note the ciliated epithelium, submucosa and muscularis and a blood vessel. The muscular bundles may be seen.

of pieces taken from the posterior wall of the specimen were sectioned, and it was found that much of the epithelium had been rubbed off or destroyed in handling. Near the neck of the diverticulum enough of the epithelium remained for study; it was certainly respiratory epithelium (figs. 6, 7 and 8). One block section was taken from the glandular thickening, and, as will be seen from the sections, the glands are typical of those of the trachea (figs. 9 and 10). Certainly the structure of the wall of the sac indicated that it was a diverticulum."

What might be the clinical importance and treatment of such a pathologic lesion? A study of the mechanics involved and of its patho-

logic process leads one to infer that it might be a contributing source of future trouble. Though apparently rare, the lesion must be excluded in diagnosing vague respiratory disturbances. From the size of the sac one is forced to believe that it might at times be distended with air. This would be especially likely if increased pressure of air was present in the trachea. In such a manner it would cause increased traction or distortion of the recurrent laryngeal nerve (fig. 2). The sac was inflam-

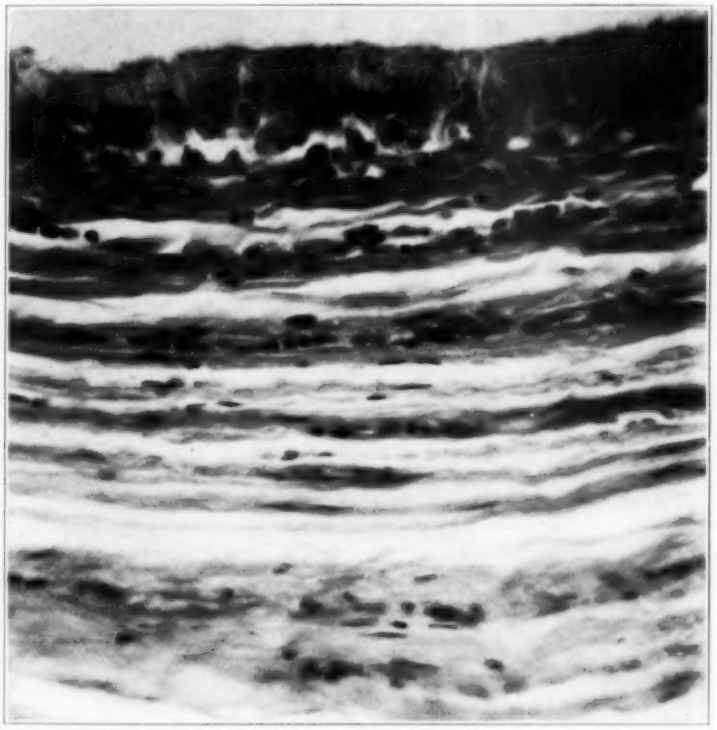

Fig. 7.—A photomicrograph (× 120) of a portion of the wall of the diverticulum. It shows the ciliated epithelium and the submucosa with a few fibroblasts within its stroma, which is indicative of a chronic inflammatory process.

matory since it was markedly adherent both to the tracheo-esophageal groove and to the recurrent nerve. From a study of the gross pathologic changes one could readily conceive the possibility of a disturbed laryngeal innervation with concomitant clinical evidence of changes in the voice. But more important were the position and size of the lesion, which served as a potential reservoir for infection, continually bathing the lower respiratory tract with purulent material, and as a persistent

source of infection of the lungs. Such a lesion has been reported as being an etiologic factor in the formation of abscess of the lung.[15]

The question might be asked why this tumor does not attain the tremendous size of an esophageal diverticulum. Peschaud [4] suggested that the peritracheal connective tissue contracts and forms an adventitious wall, thus limiting the aerial pouch. Then, too, the omohyoid aponeurosis plays an important part in limiting the dilatation. Funda-

Fig. 8.—A photomicrograph (× 240) showing epithelium and submucosa taken from a cross-section of the diverticulum. It demonstrates the nuclei of the epithelial cells and the fibro-elastic tissue of the submucosa with an occasional fibroblast.

mentally a tracheocele does not possess the same hydrostatic contents as does an esophageal diverticulum.[16]

15. Hofer, G.: Entzündliche Erkrankungen der Luftröhre und der Bronchien, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- u. Ohrenheilkunde, Berlin, Julius Springer, 1928, vol. 3, p. 9.

16. Ziegelman, Edward F.: Esophageal Diverticulum: Pulsion Type, read before the meeting of the California State Medical Society, Riverside, Calif., April 30 to May 2, 1934.

The diagnosis is difficult. The lesion must be differentiated from esophageal diverticulum and a tracheal retention cyst, since infection of the mucous glands does not always develop into a diverticulum.[17] It would seem that a diagnosis would be possible by using iodine in poppy-seed oil instilled into the trachea, with the patient in the Rose (or modified Trendelenburg) position and making roentgen studies.

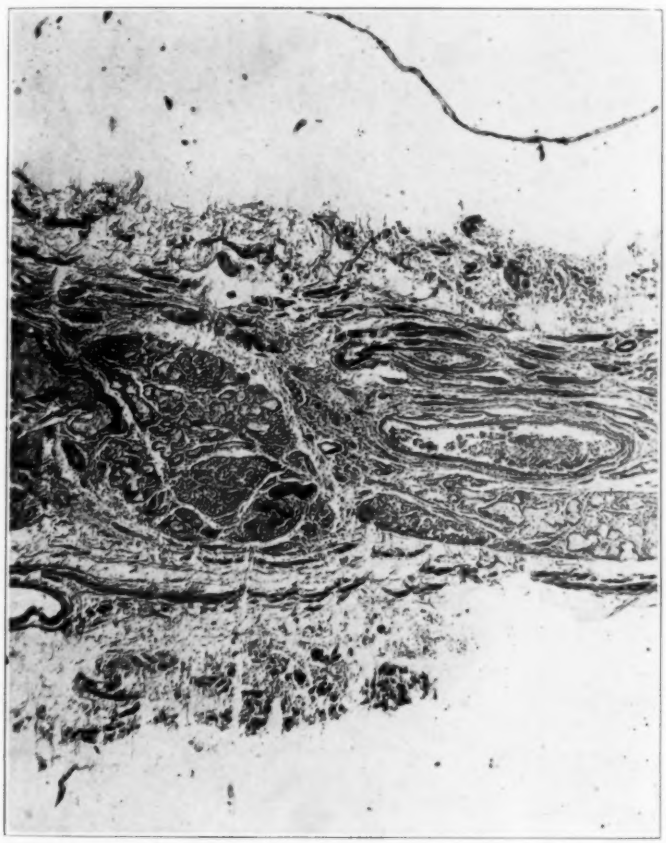

Fig. 9.—A photomicrograph (× 40) of a section through a portion of the submucosa and muscular area of the diverticulum. Note the muscular areas within the sheath and the mucous glands, especially in the lower right-hand corner. To the right of the center is seen a typical blood vessel in cross-section with its contents.

A transitory disturbance of the voice should at least make one exclude the remote possibility of such a lesion being present.

17. Maresch, Rudolf: Zur Kenntnis der aus den Schleimdrüsen der Trachea hervorgehenden, divertikelartigen Bildungen, Wien. klin. Wchnschr. **21**:824, 1908.

Extirpation of the sac by the method applicable to esophageal diverticulum seems logical. The danger of a fistula would have to be considered and likewise injury to the recurrent laryngeal nerve. This should be overcome by a free dissection of the sac and amputation near the trachea, using Lembert stitches for inversion and reenforcing with an added row of stitches in the soft parts. The safety precaution of

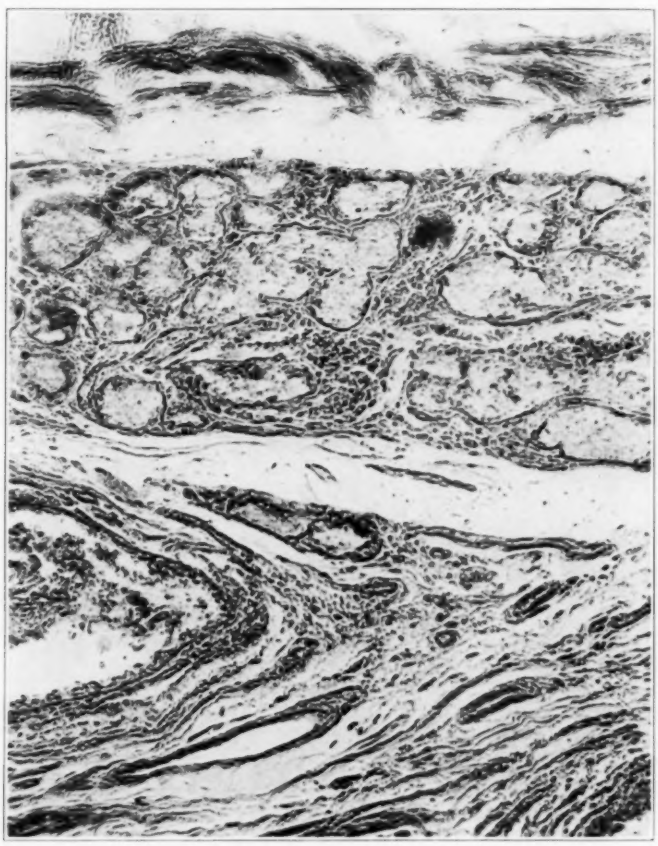

Fig. 10.—A photomicrograph (× 160) of a section through a portion of the wall of the diverticulum. It shows clearly the mucous glands of the tracheal wall. By using a hand lens the nuclei in the cells of the glands can be readily seen. To the left a portion of a blood vessel can be seen.

the two stage technic, as used in cases of esophageal diverticulum, would seem logical. The one stage method with the added use of the tracheoscope might be applicable.

If the neck and orifice are sufficiently large a return invagination into the trachea would appear to be feasible after a free dissection with

plication of the superimposed tissues and subsequent removal of the inverted sac by way of direct tracheoscopy. Tracheotomy has been advised [4] on the principle that the pressure of air would be relieved, thereby allowing the diverticulum to readjust itself.

CONCLUSIONS

1. Diverticulum of the trachea is rare.

2. In the acquired form, infection in the mucous glands of the post-tracheal wall appears to be an antecedent factor.

3. The exciting cause is probably some increase in the intratracheal pressure of air.

4. A diverticulum may attain such size as to manifest clinical evidence.

5. It serves as a potential source of infection to the lung.

6. Surgical removal by amputation or inversion with regulation of the occupation, if necessary, would seem to be the ideal treatment.

MICROSCOPIC PATHOLOGY OF THE PALATINE TONSIL

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This paper will be concerned, first, with the more common pathologic conditions of the palatine tonsil and, second, with a correlation between the clinical history which resulted in tonsillectomy and the actual microscopic diagnosis after examination of specimens from 104 routine tonsillectomies. The work was suggested by Dr. Harris P. Mosher and was carried out at the Massachusetts Eye and Ear Infirmary.

At the Massachusetts Eye and Ear Infirmary an average of 2,200 tonsillectomies are done a year. Tonsils which in any way suggest the presence of a pathologic process other than the usual chronic infection are sent to the laboratory for microscopic examination. During the year from Sept. 1, 1933, to Sept. 1, 1934, this was done in 30 cases.

The 30 cases are divided into groups according to the microscopic observations.

The normal tonsil (fig. 1) is covered with stratified squamous epithelium which dips into the deep slender crypts. These crypts should be well open and should contain no, or only a few, cellular elements. The basement membrane is usually fairly well marked but varies considerably in different specimens. The germinal centers vary in activity, size and number according to the age of the patient. The centers are separated by septums of connective tissue.

Of course, the amount of material found in the crypts depends to some extent on how hard the surgeon squeezes the tonsil in removing it. His technic is a big factor in the amount of congestion of the blood vessels and the amount of hemorrhage into the stroma. However, I feel that these two points are among the most important aids in diagnosing infection of the tonsil.

The accurate diagnosis of varying degrees of tonsillar infection is difficult. I have used five guides: 1. The amount of cellular elements and débris in the crypts is important. These are composed mainly of lymphocytes, erythrocytes and desquamated epithelial cells. The presence of polymorphonuclear cells in the crypts and tonsillar stroma is considered definite evidence of infection. Occasionally clumps of bacteria may be seen deep in the crypts. Of great importance is the presence of plugged crypts and crypts which seem to have inadequate drainage. I feel that

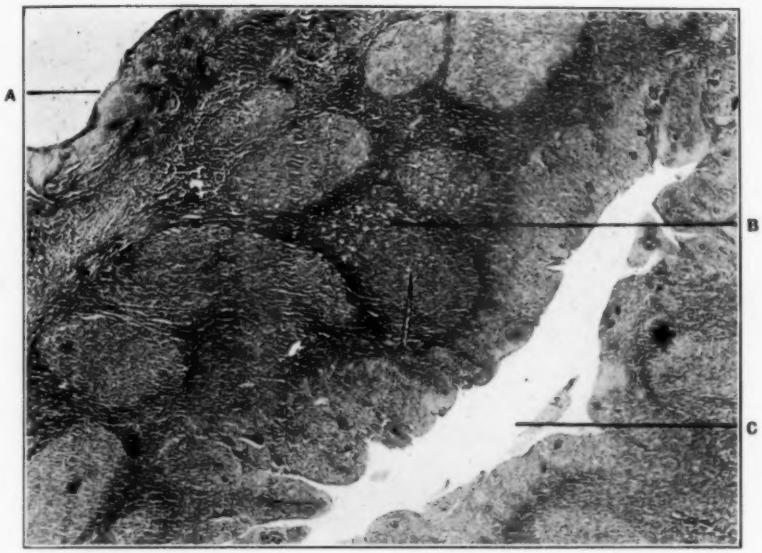

Fig. 1.—A photomicrograph of a normal tonsil from a boy 4 years old. It shows normal stratified squamous epithelium (*A*) above a well marked basement membrane. The crypt (*C*) contains no cellular elements. The germinal centers (*B*) are normal for a child of the patient's age. They show activity as evidenced by the light areas. The septums of connective tissue and the blood vessels show no abnormality. Magnification, × 30.

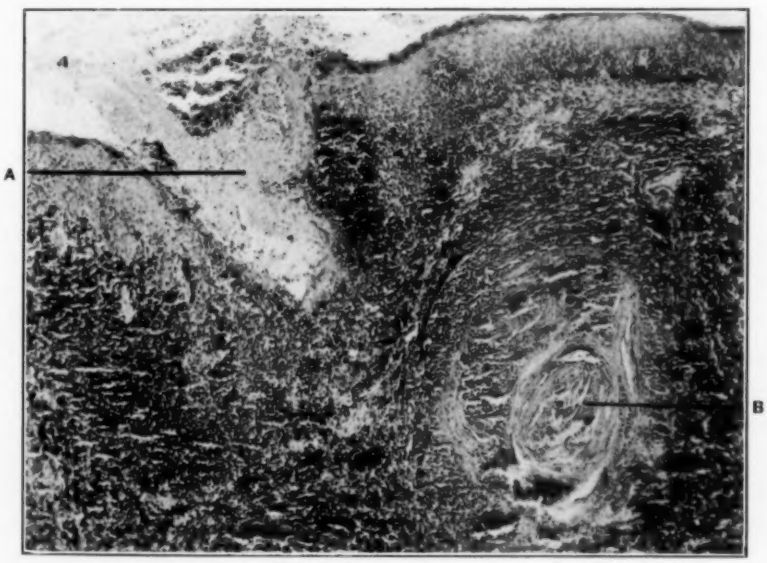

Fig. 2.—A photomicrograph of a chronically infected tonsil of a girl aged 2 years. There is a crypt filled with cellular elements (*A*), including some polymorphonuclear cells. *B* indicates a crypt which is completely plugged. The blood vessels are congested throughout, and the tonsillar stroma is almost obliterated in this area by the diffuse fibrosis. Magnification, × 70.

this is the most important point, especially since the lymphatic drainage of the tonsil is peripheral. 2. The intactness of the epithelium is a guide to diagnosis. In severe inflammation the epithelium is usually desquamated, the amount of desquamation depending on the severity of the reaction. 3. Chronic infection in any organ is accompanied by fibrosis. Hence, I take into consideration the amount of fibrosis throughout the tonsillar stroma. 4. The amount of congestion of the blood vessels and the presence of many vessels are of importance. The amount of hemorrhage depends so much on the surgeon that only a passing glance is bestowed on it. 5. The condition of the germinal centers should be noted. This, of course, depends on the age of the

Fig. 3.—A photomicrograph of the tonsil seen in figure 2, showing some desquamation of the epithelium (*A*) and marked fibrosis (*B*) running throughout the tonsillar stroma. Magnification, × 60.

patient, the centers being large, numerous and active in children and gradually fading out of the picture as the patient grows older. But one should look for abnormalities of size and number.

Figures 4 and 5 are photomicrographs of a tonsil from a man 30 years old. Figure 4 is of interest on account of the cartilage which may be seen in the capsule. This is of no particular clinical importance and merely serves to annoy the person cutting the section. Figure 5 shows a large cyst lined with squamous epithelium. Such cysts are not uncommon but become of clinical interest only when they are filled with pus, which may produce severe sore throat.

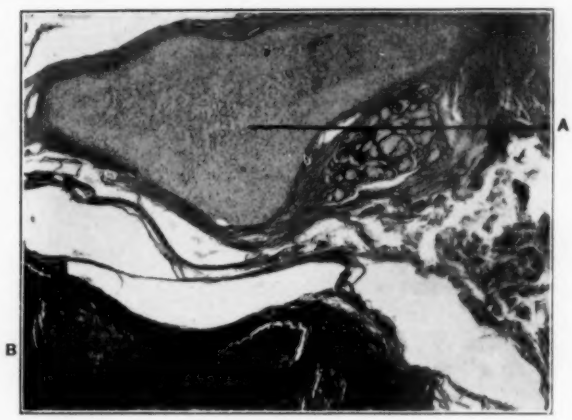

Fig. 4.—A photomicrograph showing cartilage (*A*) in the capsule of a chronically infected tonsil. *B* indicates tonsillar stroma. Magnification, × 40.

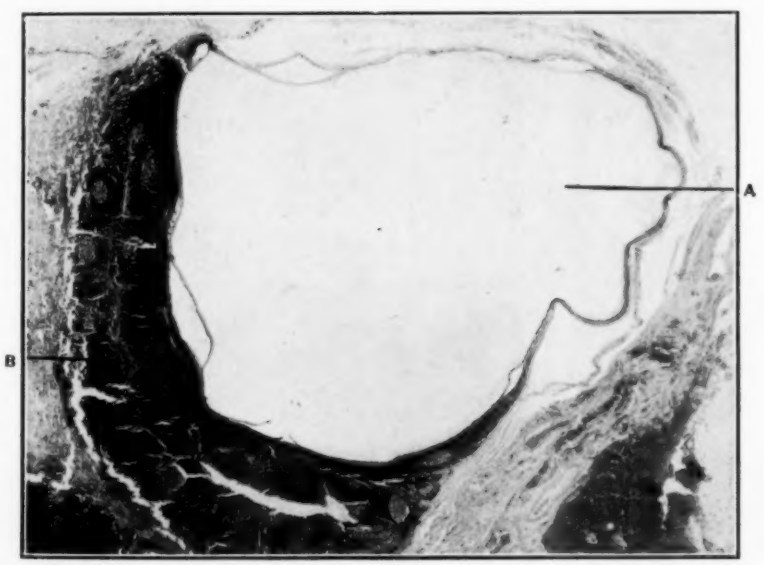

Fig. 5.—A photomicrograph of a chronically infected tonsil showing a large cyst (*A*) lined with squamous epithelium in the tonsillar stroma (*B*). Magnification, × 14.

Of the 30 suspicious tonsils examined microscopically during the past year 20, or 66⅔ per cent, showed merely chronic inflammation.

The condition next frequently noted was epidermoid carcinoma of all grades, which occurred in 13⅓ per cent of the tonsils. The section shown in figure 7 is an example of marked epithelial proliferation which has broken through the basement membranes. The cells are well differentiated and there is a tendency to epithelial pearl formation. An

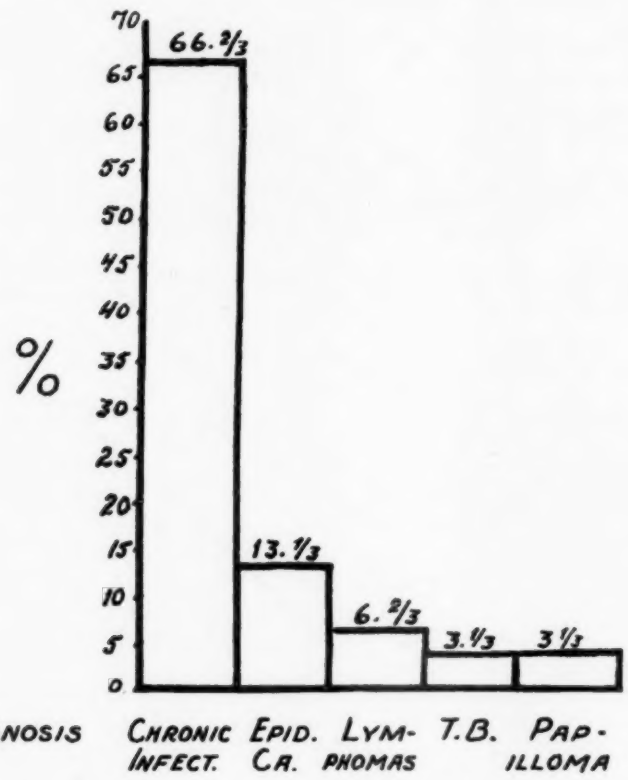

Fig. 6.—Results of microscopic diagnosis of thirty tonsils examined from Sept. 1, 1933, to Sept. 1, 1934.

epidermoid carcinoma which shows pearl formation cannot be of malignancy higher than grade 2.

Lymphoblastoma was found in 6⅔ per cent of the tonsils. As can be seen in figure 8, in a section of a tonsil removed from a man 70 years old, the chief characteristics are the uncorralled infiltration of the large undifferentiated cells, which showed many mitotic figures in the original slide, and the obliteration of all tonsillar landmarks.

Tuberculosis was present in only 1 tonsil, or 3⅓ per cent (fig. 9). Papilloma likewise appeared in only 1 tonsil, or 3⅓ per cent. The tonsil of a man 20 years old showed marked epithelial proliferation spreading out from the capsule. In figure 10 one may note the loose connective tissue body of the branching growth. The tonsillar stroma shows nothing particularly interesting and there is no evidence of a malignant process.

Fig. 7.—Photomicrograph of an epidermoid carcinoma of a tonsil from a man aged 60. The upper section (× 60) shows the marked epithelial proliferation (*B*) which has broken through the basement membrane and practically obliterated all the tonsillar stroma (*A*). The lower section (× 300) shows the well differentiated cells, the numerous mitotic figures (*C*) and the epithelial pearl formation (*D*).

The percentage of incidence as given has been with reference to a selected group of 30 tonsils which were clinically suggestive of a grave pathologic change. If these figures are translated into terms of the general run of patients, such as a physician sees in the office or in the

outpatient department, the percentage is much lower, as may be seen in figure 11.

As I mentioned earlier, in the year under consideration 2,200 tonsillectomies were performed at the Massachusetts Eye and Ear Infirmary. Only 4, or 0.18 per cent, of the tonsils were carcinomatous; in 2, or 0.09 per cent, there was lymphoblastoma, and 1 case each, or 0.045 per cent, was tuberculous and papillomatous.

I do not know why the percentages noted in this clinic were so much lower than those usually given in the literature. With regard to tuberculosis of the tonsil, Dr. Farber of the Childrens' Hospital told me that the average in that hospital is about 1,200 tonsillectomies per year.

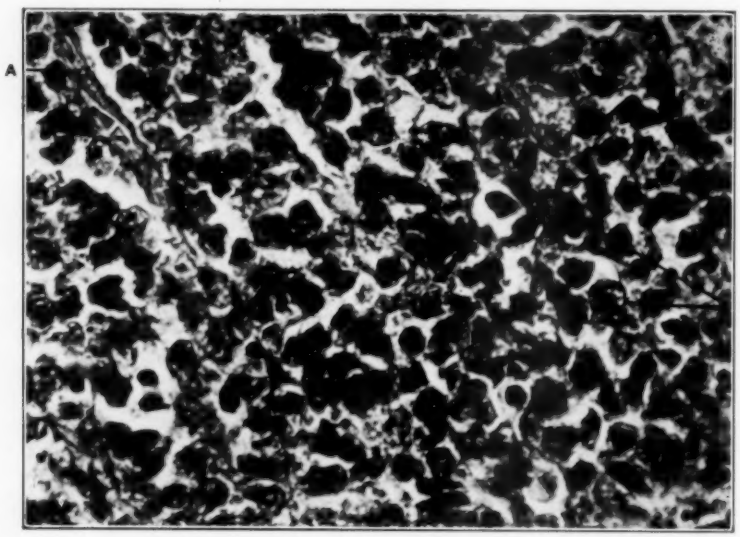

Fig. 8.—A photomicrograph of a lympho-epithelioma of a tonsil, showing the closely packed undifferentiated cells (*B*) which are without structural arrangement and which showed many mitotic figures in the original slide. Note the tumor stroma (*A*) and the obliteration of all tonsillar landmarks. Magnification, × 500.

In one year, of this number eighteen tonsils were sent to the laboratory for microscopic examination. Four of the selected tonsils were tuberculous. Hence, at the Childrens' Hospital there was a percentage of 22.2 per cent tuberculous tonsils in the selected group and 0.33 per cent in the entire group of removed tonsils. This is nearly eight times my figure.

Perhaps a few early cases are missed because routine microscopic sections are not made of all tonsils. I feel, however, that this is not the case and that it would not make an appreciable difference in the figures. The age group, as shown in figure 12, may be a factor.

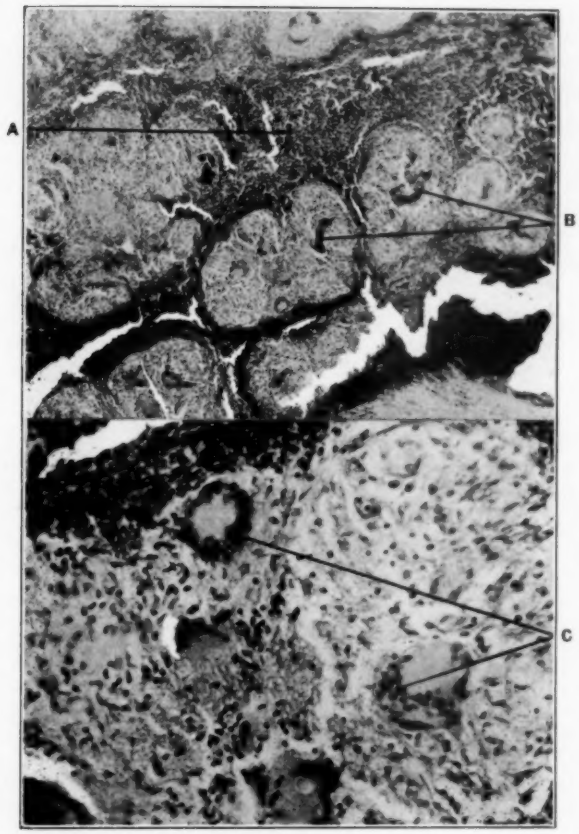

Fig. 9.—Photomicrographs showing tuberculosis of a tonsil, with numerous tubercles (*B*) in the tonsillar stroma (*A*). Note the necrotic centers surrounded by giant cells (*C*). The upper section has a magnification of 54, and the lower, of 260.

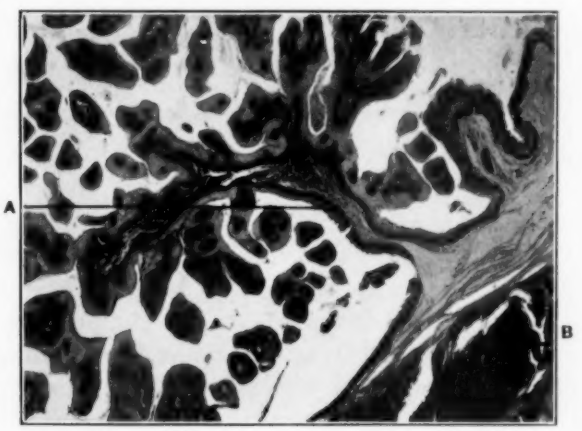

Fig. 10.—A photomicrograph of a papilloma of a tonsil, showing the epithelial branching growth springing from the tonsillar capsule (*A*). *B* indicates tonsillar stroma. Magnification × 14.

It is of passing interest that while the large majority of the male patients were in the first ten years of life the females showed nearly an equal number in each age group up to the twenty-first year. The average age of the girls was about 2½ years greater than that of the boys.

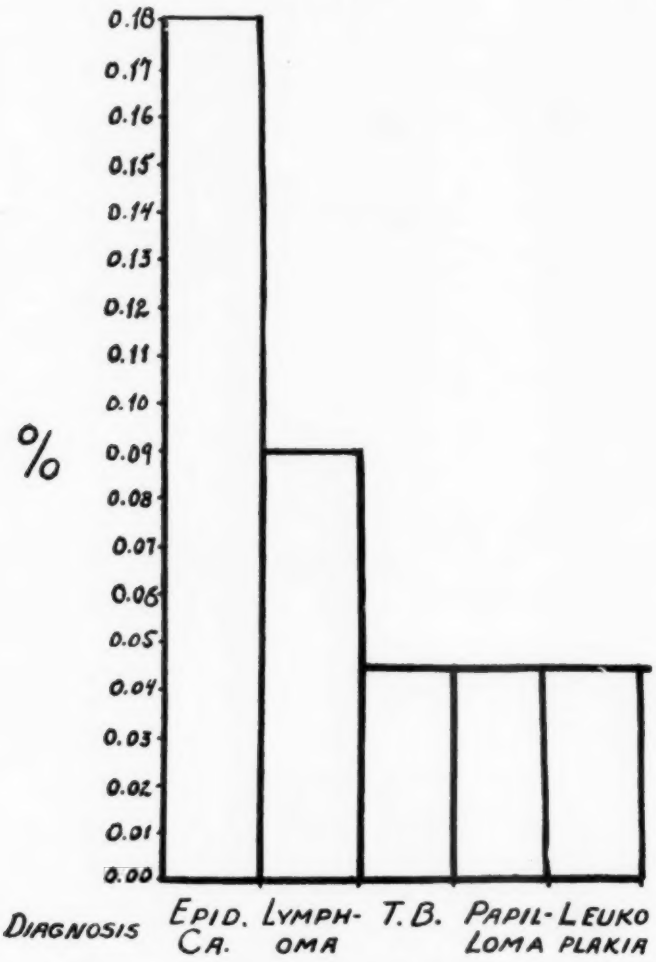

Fig. 11.—A diagram showing the percentage of pathologic tonsils, excluding those with chronic infection, in specimens from 2,200 tonsillectomies.

Recently it was decided to try to settle the old question of whether the tonsil should grace the operating table or the patient's throat. I reviewed 104 routine tonsillectomies and have attempted a correlation between the clinical history and the microscopic diagnosis. The results are shown in figure 13. As was to be expected, the largest group

of patients, 45, or 43.26 per cent, complained of recurrent sore throats and colds. Of this number, 39, or 86⅔ per cent, showed chronic tonsillar infection. The remainder were normal.

Fig. 12.—The age of 104 patients who underwent routine tonsillectomy. The left-hand diagram shows the incidence in females, and the right-hand column, that in males.

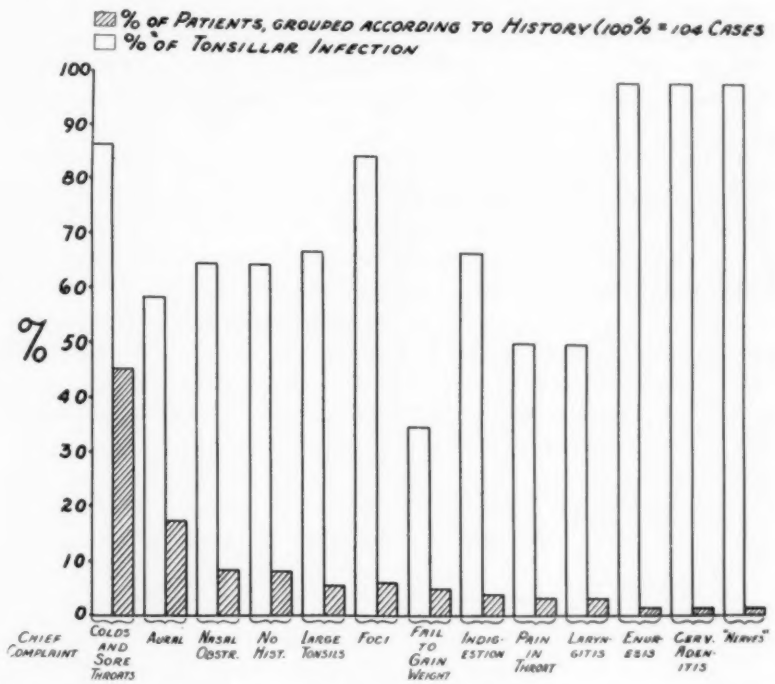

Fig. 13.—A correlation between the clinical history and the diagnosis in 104 cases.

Seventeen patients, or 16.34 per cent, underwent tonsillectomy on account of discharging ears, repeated earache or some other aural difficulty. The tonsils of only 58.85 per cent of these patients showed evidence of infection. In the 8 patients, or 7.69 per cent, whose chief

complaint was nasal obstruction there was an incidence of tonsillar infection of 62.5 per cent. Likewise, the 8 patients for whom no history was given on the clinical record had an incidence of tonsillar infection of 62.5 per cent.

The next group, 5.67 per cent of the whole, was made up of those who had been referred from the schools because of large tonsils. Of these children 66⅔ per cent had infected tonsils.

The patients in whom I was particularly interested were those whose tonsils were considered foci of infection for iritis, arthritis and so forth. This group was surprisingly small, being only 6.71 per cent of the total number, but 85.71 per cent showed infection in the tonsils.

Curiously, the same number of patients complained of failure to gain weight or complained of "indigestion," namely, vague abdominal distress and belching. Each of these groups contained 3 patients, or 2.88 per cent of the total. Of the patients with "failure to gain weight," who seemed to have the more reasonable cause for tonsillectomy, only 33⅓ per cent had tonsils which showed infection, while of the patients with "indigestion" the tonsils of 66⅔ per cent showed infection. One reassuring thing is that one of the "thin" patients who showed apparently normal tonsils has been thriving and gaining weight since the operation.

Two patients were operated on whose history recorded laryngitis, and 2 had occasional pain on either side of the throat. In these two groups there was infection in 50 per cent.

The other complaints consisted of recurrent cervical adenitis, "nerves" and enuresis, with 1 patient in each group. All showed chronically infected tonsils, making the percentage 100.

In conclusion, I have found that at the Massachusetts Eye and Ear Infirmary the incidence of carcinoma, tuberculosis and other grave pathologic conditions is very small, these conditions occurring in about 0.4 per cent of the total number of patients who undergo a routine tonsillectomy.

There is a close correlation between the clinical history which suggests some infection of the tonsil and the actual microscopic diagnosis.

PARA-ESOPHAGEAL DIAPHRAGMATIC HERNIA ASSOCIATED WITH INTRINSIC LESIONS IN THE ESOPHAGUS

REPORT OF CASES

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AND
HERMAN J. MOERSCH, M.D.
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Herniation of a portion of the cardiac end of the stomach through the esophageal hiatus in the diaphragm occurs with considerable frequency. It may be found in the course of routine examination of the gastro-intestinal tract, and it may occur without producing symptoms or may give rise to a great deal of discomfort. In many instances dysphagia is a prominent symptom.

The hernia may be the rarer type with so-called congenital shortening of the esophagus, in which a short benign stricture is responsible for dysphagia, or it may be the usual type in which a portion of the stomach herniates through the enlarged esophageal hiatus along the esophagus—the para-esophageal type of hernia.

In cases of the latter type of hernia difficulty in swallowing may be mild and intermittent or it may be so severe that esophageal obstruction is apparently complete for several hours at a time. This symptom associated with pain in the epigastrium or under the left breast frequently leads to an erroneous diagnosis of cardiospasm until roentgenoscopic examination reveals the true nature of the obstruction. Spasm at the cardia as well as pressure of the hernia on the lower end of the esophagus may be responsible for the symptoms.

Whenever a patient complains of intermittent obstruction of solid foods or liquids at the cardia associated with pain and a sense of fulness in the epigastrium, para-esophageal diaphragmatic hernia should be considered as a likely cause of the discomfort. One may be satisfied with the demonstration of a single lesion as the cause of dysphagia, but it has been pointed out previously that multiple lesions may occur in the esophagus, and unless a complete investigation of the esophagus is carried out in every case in which the patient complains of difficulty in swallowing, significant lesions may be overlooked.

In a case of para-esophageal diaphragmatic hernia in which the lesion has been demonstrated by roentgenoscopic study, it should not be assumed

From the Division of Medicine, the Mayo Clinic.

that the lesion is entirely responsible for all the symptoms, but a complete study of the esophagus should be carried out. The following report of cases will demonstrate the necessity for this investigation.

REPORT OF CASES

CASE 1.—A married woman 58 years of age was admitted for examination on Jan. 8, 1931. A carcinoma of the left breast had been removed elsewhere six years previously, and following that her general condition had been satisfactory until the onset of the symptoms that brought her to the clinic for further treatment.

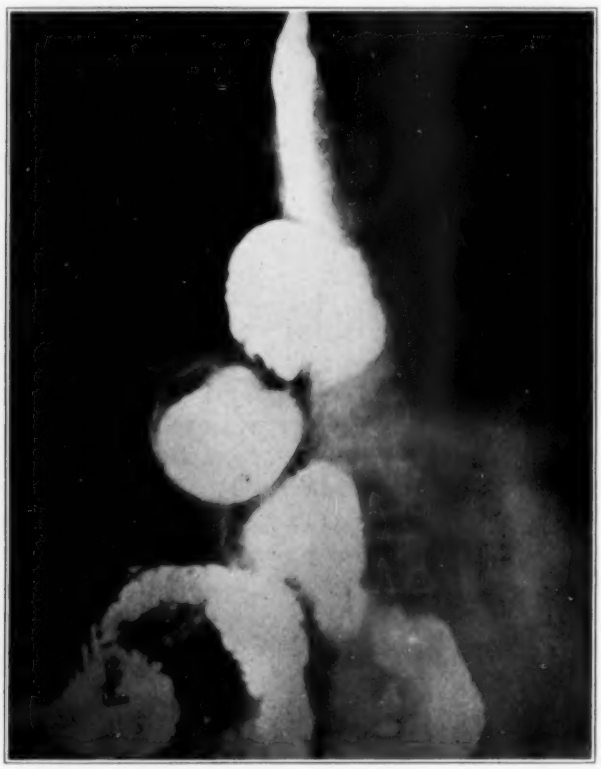

Fig. 1.—Herniation of one third of the cardiac portion of the stomach through the esophageal hiatus in the diaphragm.

Three years before admission the patient noted obstruction to the passage of solid food in the lower portion of the esophagus. This was associated with considerable pain beneath the lower end of the sternum which was severe enough to necessitate the administration of sedatives. Morphine had been used for such a long time that the daily administration of 4 grains (0.24 Gm.) of the drug was necessary to relieve the discomfort. Semisolid foods and liquids were swallowed with ease, and the patient's general nutrition was good. Roentgen examination of the esophagus and stomach revealed herniation of about one third of the cardiac portion of the stomach through the esophageal hiatus in the diaphragm (fig. 1).

There was no evidence of obstruction in the esophagus. It was believed that the hernia was responsible for all the symptoms, and operation was advised. Special study of the esophagus was not made.

At operation a third of the cardiac portion of the stomach was found to have herniated into the posterior mediastinum through the esophageal hiatus in the diaphragm. The hernia was reduced and the opening in the diaphragm repaired. An attempt was made to introduce a stomach tube, and obstruction was detected in the lower portion of the esophagus. Esophagoscopy revealed a stricture in the esophagus just above the cardia with a very small lumen. Tissue was not

Fig. 2.—Stricture in the lower portion of the esophagus and herniation of the cardiac portion of the stomach through the esophageal hiatus in the diaphragm.

removed for examination. Gastrostomy was performed, but the patient did poorly and died on the tenth day after operation. A postmortem examination was not made, and the exact nature of the esophageal obstruction was therefore not determined. The duration of symptoms suggests that it was benign.

CASE 2.—A minister 69 years of age was examined on May 23, 1927. He had suffered from hay fever for many years and had had asthma for ten years. For six years he had noted distress in the epigastrium and beneath the sternum which occurred about one-half hour after meals and lasted for an hour. Eructation of gas seemed to afford partial relief. Dysphagia had not been noted. Roentgen examination of the stomach and esophagus revealed hernia of a portion of the

cardiac end of the stomach through the esophageal opening in the diaphragm. Because of the mildness of the symptoms, the age of the patient and the associated asthmatic difficulty, operation was not considered advisable.

Two years later the patient was forced to undergo suprapubic prostatectomy for gradually progressive obstruction, from benign hypertrophy, of the prostate gland. He withstood this operation without difficulty but returned for reexamination on April 11, 1933, because of dysphagia of six months' duration. This difficulty had first been noted on swallowing solid foods and had gradually progressed in severity. He had lost 15 pounds (6.8 Kg.). On further roentgen examination

Fig. 3.—Herniation of a large portion of the cardiac end of the stomach through the esophageal hiatus in the diaphragm.

a stricture was demonstrated in the lower portion of the esophagus in addition to the hernia that had been found previously (fig. 2). The esophageal lesion was considered to be malignant.

Esophagoscopy revealed a stricture in the esophagus covered with necrotic tissue. When a specimen of this tissue was examined microscopically it was found to be inflammatory. The stricture was dilated, and ten days later a second esophagoscopic examination was made, at which time it seemed that the stricture was benign. Four dilations of the stricture were made in the succeeding seven months; at the time of the last dilation the patient was entirely free from symptoms, and he has remained well for more than a year since that time.

CASE 3.—A married woman 60 years of age was admitted for examination on June 23, 1927, because of difficulty in swallowing of one year's duration. The difficulty consisted of obstruction to the passage of solid food in the lower portion of the esophagus. When food stuck there was an associated sensation of smothering and choking which was relieved by the eructation of gas or hiccup. The patient's weight had remained about the same.

Roentgen examination of the esophagus revealed an obstruction at the cardia which was considered to be the result of cardiospasm, and a study of the stomach demonstrated a hernia of a large portion of the cardiac end of the stomach through the esophageal opening in the diaphragm (fig. 3). A no. 60 French sound was passed into the stomach without obstruction. There was temporary improvement in the symptoms; however, it was felt that the hernia was responsible for the patient's discomfort and operation was advised.

The hernia was repaired and the patient was well for five months. She then began to have a return of the symptoms which she had experienced previously. Further roentgen studies showed that the stomach was in normal position but there was obstruction at the cardia as a result of cardiospasm. Dilation of the region of spasm with a hydrostatic dilator gave the patient prompt and practically complete relief from dysphagia. Her sudden death on Nov. 13, 1934, at her home was attributed to heart disease.

CAVERNOUS SINUS THROMBOSIS OF DENTAL ORIGIN

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NEW YORK

From a study of the anatomy [1] one can readily appreciate from what varied sources the cavernous sinus is subject to attack. In a word,[2] infection anywhere about the head may, under suitable circumstances, lead to this complication.

MECHANICS

When acute bacterial infection is present about a small blood vessel (capillary or venule), transfusion of the bacterial toxins through the wall of the vessel may cause injury to the endothelial lining cells. These become swollen and blood clot is deposited in them. The thrombus at the primary site may become infected with the bacteria, or the latter may enter the vessel directly, form a colony and a thrombus form about the colony. After the initial clot has formed it may extend by successive additions up the main vessel of the area.

The pterygoid plexus drains the alveolar processes of the upper and lower jaws through the branches of the internal maxillary vein. After thrombophlebitis of the pterygoid plexus, retrograde thrombosis may take place through a small emissary vein communicating directly with the cavernous sinus or through the branch from the inferior ophthalmic vein, which in turn runs into the cavernous sinus.

A second mechanism is by dislodgment of a septic embolus from the pterygoid plexus, which can take the route from the deep facial vein to the facial vein. Then a reversed current can occur up the valveless facial vein, angular and ophthalmic veins to the cavernous sinus. Here the septic embolus, caught in the fibrous network interlocking this sinus, may give rise to thrombophlebitis of the cavernous sinus.

In the rare instances in which recovery has taken place there has been a recanalization of the thrombus and utilization of anastomosing blood vessels.

SYMPTOMS

The symptoms [3] are due to general sepsis, to metastatic foci if the blood stream becomes involved, to mechanical obstruction to the return

From the Hospital for Joint Diseases.

1. Morris, Henry: Morris's Human Anatomy, ed. 8, Philadelphia, P. Blakiston's Son & Co., 1925.

2. de Schweinitz, G. E.: Thrombosis of Cavernous Sinus, Atlantic M. J. **27**:551 (June) 1924.

3. Smith, Dorland: Cavernous Sinus Thrombosis with Notes of Five Cases, Arch. Ophth. **47**:482 (Sept.) 1918.

circulation, to interference with the function of nerves in the vicinity, and to the rapidly complicating meningitis.

The classic picture, therefore, is that of high fever, rapid pulse and general prostration. If the thrombus breaks down, metastatic abscesses appear in various parts of the body, particularly in the lungs.

The veins in the vicinity of the eye become prominent; there are increasing proptosis of the eyeball, with perhaps a slight headache, and a slight rigidity of the neck. There are palsy of the oculomotor group, with a dilated, rigid pupil and hyperesthesia in the distribution of the fifth nerve. The conjunctiva begins to protrude in yellowish folds, the patient starts to vomit, a Babinski sign may appear, the other eye becomes involved and the patient dies in coma within forty-eight hours.

DIFFERENTIAL DIAGNOSIS

The picture [3] when fully developed is hardly in need of a differential diagnosis. However, the following diseases should be considered: cellulitis of the cheek accompanied by edema of the eyelid (case 3), orbital cellulitis, infection of the accessory sinuses, exohpthalmic goiter and tumors of the orbit, optic nerve and lacrimal gland.

SUMMARY

We have, therefore, in thrombosis of the cavernous sinus a disease in which the pathologic changes, progress of the infection and symptoms can be readily explained and understood from the anatomy of the region, a disease which may follow trivial infections in the area drained by the sinus.[4] The staphylococcus is usually the organism concerned, and meningitis follows so closely after the ocular symptoms develop that septic thrombosis of the cavernous sinus is generally conceded to be a nonsurgical complication.

ILLUSTRATIVE CASES [5]

The following summaries of cases and photographs are used for illustration.

CASE 1.—J. A., a boy aged 4 years and 11 months, was admitted to the hospital on Jan. 9 and died on Jan. 24, 1933. Ten days before admission he had an attack of influenza from which he made an uneventful recovery. Three days before admission he complained of toothache about the left upper incisors. On January 8 it was noted that the gums about this area were oozing blood and a small amount of pus, and that the upper lip was swollen.

4. Dixon, O. J.: Pathological Examination in Cavernous Sinus Thrombosis as a Guide to Diagnosis, Prognosis and Treatment, J. A. M. A. **87**:1088 (Oct. 2) 1926.

5. Dr. Adolf Berger and Dr. Philip Grausman gave me permission to use the case histories, and Dr. Sheldon A. Jacobson, to use the autopsy report.

The past history was negative except for paracentesis of the ear drums at the age of 2.

On admission, the child appeared acutely ill but intelligent and cooperative. The most noticeable feature was a large, soft, red swelling of the lip extending from one ala of the nose to the other. About the gingival crevice of the left upper central incisor (which was loose) there was a discharge of blood, and the mucobuccal fold was obliterated by the swelling in the tissue of the lip. The pupils were equal and reacted to light and in accommodation. The right ear drum was perforated, and both drums were moderately congested.

On admission, a diagnosis of alveolar abscess with secondary cellulitis of the upper lip was made.

A reference to the photograph gives a more vivid account of what occurred in a week's illness than a lengthy description could. Metastatic abscesses appeared in various parts of the body, including the face. Incisions were made to no avail, and the child died in coma.

Fig. 1 (case 1).—Photograph taken after one week's illness.

CASE 2.—M. S., a woman aged 50, was admitted to the hospital on April 9 and died on April 19. She complained of swelling of the left side of the face and pain in the lower jaw of that side, inability to open the mouth and difficulty in swallowing. She gave a history of having had swelling of the left side of the face one month ago and the third left lower molar was extracted one week previous to hospitalization. This was followed almost immediately by swelling of the lips, cheek and lower eyelid, which continued to increase until she presented herself at the hospital, desperately ill, with a temperature of 106 F. She had repeated chills that night.

Local examination revealed nonindurated swelling of the entire left side of the face, more marked beneath the mandible at the angle of the jaw. There was marked difficulty in opening the mouth and in swallowing. The left lower molars were missing. There was edema of the soft palate and peritonsillar region.

The x-ray picture made at this time was suggestive of osteomyelitis of the ramus on the left side.

On April 12, an extra-oral incision was made and a probe inserted to the lingual surface of the bone. On April 14, an intra-oral incision was made on the anterior border of the left ramus.

The patient was now stuporous, irrational at times, and complained of severe headache and blurring of vision of the left eye. She was vomiting, and rigidity of the neck was present, with slight edema of the right eyelid.

By April 18 the picture was unfortunately classic. The right eye was closed, and there was marked proptosis of both eyeballs. The conjunctiva protruded in loose folds beneath the eyelids. The right eyeball was fixed, and the pupil did not react to light. There were marked rigidity of the neck and pain on attempted flexion. The mouth could be opened only slightly, and there was free discharge of pus from the retromolar incision. The abdomen was tender to touch. A Babinski sign was present on the right.

Fig. 2 (case 2).—Photograph taken twenty-four hours before death.

The patient died on April 19, of pulmonary edema. (A blood transfusion was given on April 19.)

Autopsy.—The organs of the neck were not removed. The tissue about the left mandible was thickly infiltrated with pus. Much of it was necrotic in appearance. The bone between this area and the middle cerebral fossa seemed somewhat infiltrated with pus. Considerable quantities of pus infiltrated the tissues around the path of the carotid artery and jugular vein on the left. The pituitary fossa and surrounding soft tissues were thickly infiltrated with pus. Microscopic section showed a purulent exudate about the pituitary gland. The pars nervosa was largely necrotic, and there was an area of necrosis in the pars glandulosa. The left cavernous sinus was thrombosed and contained pus; the right contained a fresher, apparently bland, thrombus. Other sinuses were distended with postmortem thrombi.

The brain was normal in size and shape. The gyri and sulci were normally developed. The surface veins appeared considerably distended, particularly those on the left. The base of the brain in the angle between the frontal and temporal lobes showed a collection of pus in the leptomeninges, apparently infiltrating the

brain substance for a very short distance. The nuclei showed nothing noteworthy. Microscopic section showed practically no penetration of pus into the brain substance. The ophthalmic veins on the left were thrombosed as was the left jugular vein.

Several pieces of bone from the sinus regions on the left side and a piece of the left lower mandible were taken. They showed osteomyelitis.

CASE 3.—S. P., a man aged 24 years, was admitted to the hospital on July 13 and discharged on July 22, 1933. He complained of swelling of the left side of the face, chilliness and headache. He gave a history of having had the left lower first molar extracted one week previously, which was followed by swelling of the left side of the face. Incisions were made extra-orally and intra-orally. These incisions failed to relieve the swelling. On the day of admission the patient noticed closing of the left eye.

Fig. 3 (case 3).—Photograph taken on admission to the hospital.

On admission, he appeared acutely ill, with swelling as seen in the photograph. The swelling was brawny and indurated and extended from the left ear and angle of the jaw to the midline of the face. The skin over the swelling was reddened and tender. No fluctuation was present. The left eye was closed, but when the lid was lifted the pupil was found to be equal in size to that on the right. The conjunctiva was reddened at the extreme left, but the eyeball moved normally in all directions. There was no involvement of cranial nerves. The only other finding of note was an enlarged and somewhat tender spleen. A roentgenogram revealed the presence of retained roots of the left lower first molar but was otherwise negative.

On July 14, an incision was made in the mucobuccal fold and a large amount of pus evacuated.

On July 15, examination of the fundus failed to reveal evidence of exudation, hemorrhage or vascular change. The same evening an incision was made over the

anterior edge of the parotid gland and about 5 cc. of pus evacuated. A grooved director was inserted toward the malar bone and a large amount of foul-smelling, thin pus evacuated.

From this time on there was an uneventful recovery.

In the third case, in which recovery took place (not a case of cavernous sinus thrombosis, but one of local infection), the following points should be noted: 1. The temperature was not sustained. 2. There was early localization of pus. 3. No classic ocular signs were noted. 4. No toxic granules were present. Toxic granules are basophilic cytoplasmic granules appearing in the polymorphonuclear leukocytes under certain conditions of infection.[6]

In the fatal cases there was present throughout the period of illness a toxic degenerative index of from three to four plus, which indicated a serious prognosis.

SUMMARY

A brief résumé of the anatomy, pathologic changes and symptoms of cavernous sinus thrombosis is given, with the histories of two cases of dental etiology and of another case for differential diagnosis. The possible significance of the toxic degenerative index in prognosis is indicated.[6]

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6. Sutro, C. J.: Cytoplasmic Changes in Circulating Leukocytes in Infection, Arch. Int. Med. **51**:747 (May) 1933.

IONIZATION AS A PROLONGED PALLIATIVE IN VASOMOTOR RHINITIS

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The object of the following presentation is to call attention to a method of ionization which has a pronounced and lasting palliative effect in cases of vasomotor rhinitis.

NATURE OF VASOMOTOR RHINITIS

The factors underlying vasomotor conditions of the nose are not sharply defined. Some authors have assumed that vasomotor rhinitis is a local manifestation of a constitutional allergic state; others have expressed the belief that it is a symptom of some metabolic, endocrinic or nervous disorder. Whichever view is correct, it is generally agreed that this type of rhinitis is not a disease entity, even though substantial proof is still lacking. The nasal condition itself is not a true inflammation but an edema of the membranous tissues characterized by eosinophilic infiltration. As this manifestation is the predominating phenomenon for which patients seek relief, it is clear that rhinologists must pay attention to it in addition to giving whatever general treatment they deem indicated to influence the underlying constitutional factor or factors.

Diagnostic difficulties are not limited to discovering the constitutional factors, for not infrequently the condition is masked by sinusitis or other nasal involvement. Lindsay and Walsh [1] have suggested a simple and effective diagnostic measure in the presence of such a complication. According to these authors, the presence of eosinophils in nasal secretions is conclusive for the diagnosis of vasomotor rhinitis. In the presence of a complicating infection the ratio of eosinophils to neutrophils is lowered, while in infections without coexisting vasomotor rhinitis the eosinophil count in the nasal secretions never exceeds 1 per cent.

REVIEW OF THERAPY OF VASOMOTOR RHINITIS

While the incidence of true hyperesthetic rhinitis is probably not as large as it is commonly believed to be, it nevertheless presents a

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1. Lindsay, J. R., and Walsh, T. E.: Nasal Secretions: The Value of Cytologic Examination to the Rhinologist, Arch. Otolaryng. **17**:783 (June) 1933.

chapter in rhinology which from a therapeutic standpoint leaves much to be desired. Every experienced physician knows that of the many and varied methods of treatment that have been tried none has proved satisfying either to the patient or to the rhinologist, and while one finds in the literature an occasional report of a cure, the cure has been found wanting when put to the test. Certainly there is as yet no true curative measure.

If one assumes an allergic basis for the disease it is rational to search for the offending allergen, to remove it from contact with the afflicted person and to effect specific immunization. Theoretically sound as this is, I have not observed the degree of success claimed for it by some enthusiasts. As a matter of fact, failures have compelled me to resort to nonspecific measures, some of which occasionally are of benefit, but which more often fail to abate even slightly the distressing symptoms.

The numerous forms of nonspecific treatment may be regarded as properly grouped in the following order:

I. Systemic
   - A. Metabolic
   - B. Pharmacal
   - C. Endocrinic
   - D. Dietetic
   - E. Residential

II. Topical
   - A. Medical
     1. Medicinal applications
     2. Irrigations
     3. Sprays
     4. Injections into the turbinals
     5. Chemical cauterization of the inferior turbinals
   - B. Surgical
     1. Injections into the sphenopalatine ganglion
     2. Removal of septal obstructions
   - C. Physical
     1. Massage
     2. Diathermy
     3. Phototherapy
     4. Galvanization
        - (*a*) Galvanocauterization of sensitive areas of the nasal mucosa
        - (*b*) Electrolysis of the inferior turbinals
        - (*c*) Ionization

INTRANASAL IONIZATION

Of all the procedures enumerated, which are too well known to require comment, I shall restrict my consideration here to ionization, not because it is new, for it has already been employed for certain other

nasal conditions, but because it has but recently been purposively utilized in the treatment of vasomotor rhinitis.

As is seen from the tabulation, ionization is based on the utilization of the galvanic current. According to St. Clair Thomson,[2] electrolysis of the inferior turbinals was resorted to by Creswell Baber as early as 1898, to overcome nasal hydrorrhea. More recently Norrie [3] satisfactorily applied this method to reduce the inferior turbinal bodies.

As regards ionization proper, the technic of which will be given, Cottle and I [4] employed it several years ago in the treatment of simple chronic rhinitis. The effects of this method led me to give it a thorough trial in the treatment of vasomotor rhinitis. As the results have been more satisfying than I had hoped for [5] and as similar successes have been reported by Warwick [6] and Alden [7] in the United States and by Démétriades [8] and Franklin [9] abroad, it cannot be assumed that all were merely accidental or coincidental.

No one can claim that intranasal ionization is a cure for vasomotor rhinitis. However, the palliative effects are so gratifying as to justify all rhinologists in availing themselves of the method in order to afford their patients relief for a comparatively long period.

TECHNIC OF INTRANASAL IONIZATION

Friel,[10] Campbell [11] and Warwick [6] have described technics, each possessing certain advantages but also certain drawbacks. My own technic, which has been tried in more than a thousand treatments, has the advantage of simplicity, to obtain which I devised the apparatus shown in figure 1.

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2. Thomson, St. Clair: Diseases of the Nose and Throat, New York, D. Appleton & Company, 1927.

3. Norrie, F. H. B.: Enlarged Turbinals: Treatment by Zinc Electrolysis, J. Laryng. & Otol. **41**:87 (Feb.) 1926.

4. Hollender, A. R., and Cottle, M. H.: Recent Advances in the Treatment of Nasal Accessory Sinus Disease, Eye, Ear, Nose & Throat Monthly **5**:575 (March) 1926.

5. Hollender, A. R.: Intranasal Ionization: Its Fundamental Aspects and Clinical Value, Arch. Phys. Therapy **5**:581 (Oct.) 1934.

6. Warwick, Harold L.: Treatment of Hay Fever and Its Allied Conditions by Ionization: Preliminary Report, Laryngoscope **44**:173 (March) 1934.

7. Alden, Arthur M.: A Year's Work with Ionization in the Treatment of Hay Fever, Larygnoscope **44**:741 (Sept.) 1934.

8. Démétriades, T. D.: Zur Behandlung der vasomotorischen Störungen der Nase durch Iontophorese, Monatschr. f. Ohrenh. **61**:524 (May-June) 1927.

9. Franklin, P.: Intranasal Treatment by Ionization in Hay Fever, Vasomotor Rhinitis and Ozena, Brit. M. J. **1**:751 (April 23) 1932.

10. Friel, A. R.: Zinc Ionization in the Treatment of Suppuration of the Maxillary, Sphenoid and Frontal Sinuses, Brit. M. J. **2**:204 (Sept. 10) 1921.

11. Campbell, A.: Ionization in the Nose: New Technic, J. Laryng. & Otol. **43**:98 (Feb.) 1928.

For the benefit of those readers who may not be familiar with the galvanic current I may say that its source is either a battery of electric cells or the commercial current for lighting. If a community is provided with only a lighting main with an alternating current, a converter has to be interposed between the city current and the apparatus in order to feed a direct current into the appliance. The essential property of the galvanic current for this purpose is absolute smoothness of flow. Whatever appliance is used must be provided with a suitable rheostat to permit a gentle increase of the current or an equally gentle reduction to prevent faradic-like "shocks" and with a milliamperemeter to measure the intensity of the current. The entire set needed for good work is as simple as it is inexpensive; complicated and costly apparatus are altogether unnecessary.

The current is led to the patient by two insulated wire cords, one connected with the positive and the other with the negative post of the apparatus. As

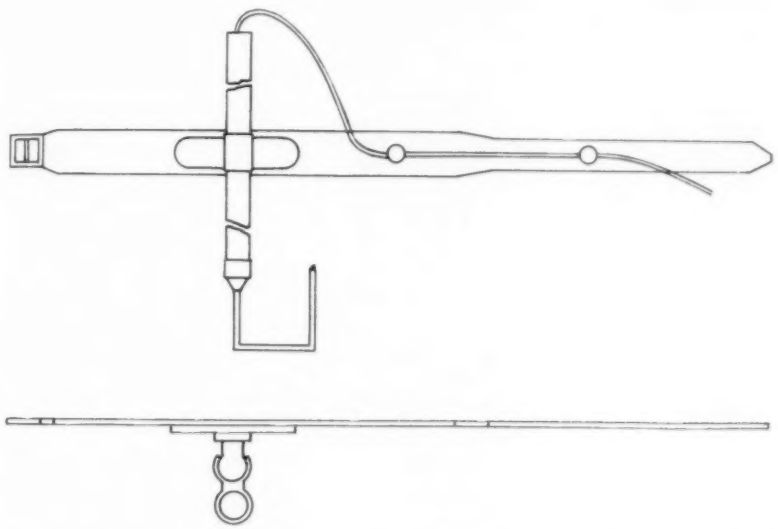

Fig. 1.—A schematic drawing of the head-band which simplifies the application of the positive pole of the electrode to the nasal packing.

topical application to the nose is made with the positive pole, the circuit with the patient is closed (or made) by connecting the negative cord to a heavy felt-covered metal electrode with 4 by 7 inches (10 by 17.5 cm.) of surface which, after being soaked in warm water, is fastened around the forearm or to the nape of the neck.

Before the positive electrode is applied to the nose one of the nasal chambers is packed with ribbon gauze which is thoroughly moistened with the so-called electrolyte. This is a solution of copper, zinc or some other metallic chemical. In my hands the best results have been obtained with an aqueous solution of zinc sulphate. Friel employs a preparation consisting of zinc sulphate, 5 Gm., glycerin, 60 cc., and water to make 1,000 cc. Friel, accordingly, makes use of a five-tenths per cent solution of zinc sulphate, which some workers have preferred to weaken by dilution and others have preferred to strengthen. As a general rule a 1 per cent solution of zinc sulphate has proved satisfactory, in which case the

zinc sulphate content of the formula will be 10 Gm., or 150 grains, in 2 ounces of glycerin and 1 quart of water.

The positive electrode in position during the process of treatment is shown in figure 2. The electrode is a piece of flexible zinc wire fastened in a suitable holder and fixed by means of a universal joint which is mounted on a leather head-band. The band is made so that it can be adjusted to a head of any size. When the band is tightened the electrode holder is in line with the nose, necessitating only a bending of the flexible wire into the wet nasal packing, caution being exercised to avoid contact with the nasal mucosa. After good contact is established at both poles the current is switched on while the handle of the

Fig. 2.—The electrodes in position during the process of ionization of the right nasal chamber. The curved zinc wire is in firm contact with the wet packing in the nose.

rheostat is at the zero end, that is to say, at a point where no current can pass. The handle is then pushed along the rheostat slowly and steadily until the indicator on the milliamperemeter shows the desired milliamperage. The dose varies, an average of 5 milliamperes being sufficient for children and 10 milliamperes for adults. For the latter the dose may be increased gradually to 15 milliamperes. The duration of the treatment depends largely on the milliamperage used. Generally if a current strength of 10 milliamperes is well tolerated the treatment should be applied for fifteen minutes, giving a total of 150 milliampere-minutes—a term applied to the total obtained by multiplying the number of milliamperes by the number of minutes the current is applied. In the

large majority of cases one ionization to each nasal chamber proved adequate, even in severe cases of true vasomotor rhinitis. If a repetition of the treatment is required it should be undertaken only after a lapse of from one to two weeks. As many as four ionizations to each nasal chamber should be given before failure is acknowledged.

One important factor which enters into the correct technical application of intranasal ionization is the body resistance, but if the following rule is adhered to no difficulty will be encountered: The current is gradually raised from 0 to 3 or 4 milliamperes and held at that intensity for about three minutes. After this interval the current is very gradually increased to the maximum of comfortable tolerance.

EFFECTS OF INTRANASAL IONIZATION

Without entering into a discussion of the physiologic and electrochemical effects of ionization it will suffice for the immediate purpose to point out that certain reactions are felt by the patient and a description of these should be imparted before treatment is given. A pronounced metallic taste and profuse salivation occur during the treatment, and symptoms similar to those of acute coryza appear shortly afterward. Examination after the removal of the nasal packing discloses a grayish coating of the entire nasal mucosa, but this coating disappears after a few days. A slight exudate which forms during the first few days either loosens and is blown out or can be removed if it produces any marked discomfort. Although all local reactions in many instances do not disappear before a week, subjective improvement is reported by the patient within a few days.

As was pointed out earlier, I have given in the course of the past twelve years more than a thousand treatments, and I have yet to observe any deleterious effects. This experience coincides with at least an equal number of like treatments administered by several colleagues.

CLINICAL EXPERIENCE

In a recently reported series of thirty-two cases of undoubted vasomotor rhinitis treatment was given in ten during the year 1932. Five (50 per cent) of the patients were definitely relieved of their symptoms after intranasal ionization by the technic described here. These patients have had no symptomatic recurrence. Seven patients were treated during the year 1933, and of these all were relieved to some degree, four being free from any nasal manifestations at the time of writing. During the first six months of 1934 I treated fifteen patients, of whom eight reported at regular intervals complete absence of symptomatic recurrence, the remainder being only partially benefited. It is, of course, too early to include this last group in a final report, but the results certainly show that zinc ionization produces at least prolonged palliation of symptoms.

Almost all the patients showed a typical clinical and pathologic picture of vasomotor rhinitis. It will therefore suffice for the purpose of illustration to report a few of the cases in which there was improvement, selected at random from office and clinic records.

REPORT OF CASES

CASE 1.—J. W., a man, aged 38, a clerk, had had symptoms of vasomotor rhinitis for one year. Submucous resection was performed in 1930 with good results so far as removal of the mechanical obstruction was concerned. The patient's general physical status was good. The onset of allergic symptoms occurred in February 1931. The type of allergy was undetermined. Two ionization treatments were given in each nasal passage, two weeks apart, in August 1932. There has been no recurrence of symptoms up to the time of writing.

CASE 2.—L. S., a man, aged 26, a merchant, had had symptoms of vasomotor rhinitis for two years. The type of allergy was undetermined in spite of numerous diagnostic skin tests. Cauterization of the inferior turbinals with galvanic cautery gave slight relief for a short period. There was no general physical impairment. The onset of nasal manifestations occurred in July 1930. The nasal passages were ionized in July 1932 with complete arrest of symptoms.

CASE 3.—A housewife, aged 30, had had symptoms of vasomotor rhinitis for five years. Anatomically the nasal condition was good. Diagnostic tests demonstrated sensitivity to house dust. Desensitization was ineffective in bringing about relief. Ionization was performed and repeated once in July 1932. Relief of symptoms occurred within three weeks, and there had been no recurrence when the patient last reported in December 1934.

CASE 4.—R. L., a housewife, aged 45, had nasal vasomotor disturbances for three years. Diagnostic tests showed sensitivity to various foods, the exclusion of which from the diet failed to improve the patient's condition. The physical status otherwise was poor, for the patient required gynecological surgical intervention. Ionization was performed in May 1933, producing about 70 per cent improvement. Repetition of treatment was refused because the patient claimed that she was more comfortable than she had been before ionization.

CASE 5.—R. T., a youth, aged 13, a student, had nasal blockage, sneezing and lacrimation of one year's duration, the onset following a severe cold after swimming. The nasal sinuses were normal at the time of examination in January 1934. The parents refused to permit tests for allergy, but resorted to all sorts of medical and nasal treatment. Sneezing was relieved on the internal administration of small doses of ephedrine hydrochloride. Ionization was performed on Jan. 20, 1934, producing complete palliation of the symptoms. The patient has reported at monthly intervals since, and has stated that there has been no paroxysm of sneezing and that the nasal passages have been clear. His general health has improved, and there has been a considerable gain in weight.

SUMMARY AND CONCLUSIONS

1. It has not been definitely established whether vasomotor rhinitis is a local manifestation of a constitutional allergic state or a symptom of a metabolic, endocrine or nervous disorder.

2. True vasomotor rhinitis masked by infection can be diagnosed by determination of the eosinophils in the nasal secretions.

3. Pathologically, vasomotor rhinitis is not a process of true nasal inflammation but an edema with eosinophilic infiltration.

4. The theoretically rational method of management of vasomotor rhinitis directed to the removal of the recognized allergen and specific immunization has not proved to be invariably successful.

5. Nonspecific therapeutic measures, both local and systemic, have occasionally proved of benefit but more often have resulted in failure.

6. Ionization, which is a comparatively new physical therapeutic agency, has produced striking and prolonged palliation of symptoms in a large number of cases of vasomotor rhinitis.

7. The technic of intranasal ionization has been simplified by an apparatus which I have devised.

8. Ionization of the nasal passages can be carried out with any other desired method of therapeutic management of vasomotor rhinitis.

9. Intranasal ionization produces comparatively mild reactions for a short period, but is an absolutely safe procedure devoid of any risk or ill effects.

10. The simplicity of technic, the favorable results and the safety of intranasal ionization fully justify its wider clinical employment in the treatment of vasomotor rhinitis.

30 North Michigan Avenue.

IONIZATION

CIRCUIT PLANS FOR AN INEXPENSIVE UNIT

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Ionization is the splitting or ionizing of a solution by the passage of a direct current. The acid and alkaline (or radical) ions are drawn to opposite poles. For treatment, the method presupposes the drawing into the tissue of chemicals seeking their pole. In practice, the methods are like those of electroplating. A solution is placed on a part by means of a wet dressing, or is poured into a cavity, and an electrode is attached; a so-called neutral electrode is placed on a nearby convenient part. The current is turned on gradually; the solution is split and one of its elements seeks the distant pole, thus passing into the tissue.

A supply of direct current plus an ammeter and electrodes (one for the ear and one for the arm) comprise the essential features of the apparatus used. Switches, a pole charger and a signal light are desirable additions. A 2 per cent solution of zinc sulphate is the electrolyte.

The supply of direct current may be had in three ways: 1. If the house current is direct it may be attenuated (I advise against this). 2. If the house current is alternating a rotary converter or motor generator may be used (I advise against this). 3. A set of dry cells is the best source of current, and 90 volts of B battery dry cells is sufficient.

The ammeter is a milliamperemeter of the best quality, reading from 15 to 30 milliamperes in large scale.

The attenuator must be smooth, providing a continuous contact. It is most important that it not be the button or coiled resistance wire type.

All terminals must be rugged and dependable; soldering is desirable in all permanent wiring. The point of this is to avoid a break in the contact while the apparatus is in use.

The arm-band for the neutral electrode must contain enough gauze or cotton so that when moistened it will not dry out within from five to ten minutes.

The ear electrodes must likewise be selected or planned with care. For children as well as for adults it is well to fix the electrode to an insulated leather head-band, for otherwise movement may cause it to drop out of the ear.

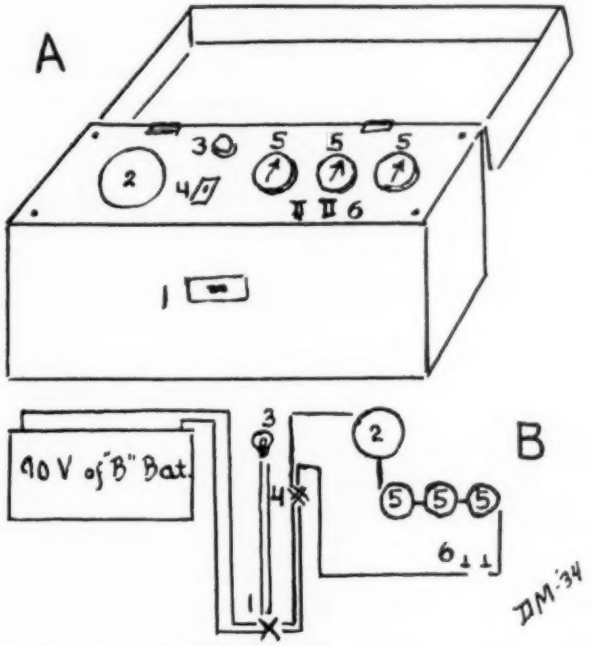
Any break in the current while the apparatus is on causes a shock.

The anode pole is attached to the positive ear electrode and the cathode to the moistened arm-band. It is essential that the operator

Read before the Philadelphia Laryngological Society, Dec. 4, 1934.

always test the polarity before starting, never depending on a previous set-up. This is done by placing both cord terminals in a glass of water. Bubbles arise from the cathode pole.

Before the positive electrode is inserted, the ear is thoroughly cleansed by douching and is filled with the solution of zinc sulphate. The current is then turned on slowly and is turned off slowly when the course is finished. In that way dizziness and nausea are avoided.



A is a diagram of an inexpensive ionization unit, showing: *1*, switch; *2*, milliamperemeter; *3*, signal light; *4*, pole changer; *5*, attenuators, and *6*, terminals. *B* is a diagram of the circuit plan; *5* indicates a rheostat with a capacity of 200,000 ohms.

Tolerance varies with the individual patient and is a rather whimsical thing. I have treated children who could tolerate 6 milliamperes. The average child or adult will not tolerate more than 2.5 milliamperes. Three minutes seems to be a satisfactory period to most workers who use this method as part of the routine.

USES IN TREATMENT OF THE EAR

I believe that ionization is useful in the treatment of many simple cases of chronically discharging ears. The discharge will cease with this method when other methods have failed. It is well to use the other

methods on a series of controls or on the patients before treatment with ionization before forming an opinion of the value of the ionization method.

Ears in which there are polyps, cholesteatoma and granulations are seldom made dry but are often improved noticeably. Ears infected with Monilia or other fungi are never improved whether the discharge appears to be simple catarrh or not. This is most important; one should look for mold by smear in all cases. One will most often find it in the foul discolored discharge. The presence of fungi is not rare.

After mastoiditis with a continuance of the discharge ionization is recommended. There should be no attempt to substitute ionization for operation on the mastoid in acute or chronic cases or in cases in which the condition clears up with each ionization treatment but recurs.

Ionization should be looked on as a valuable adjuvant method to a rational series of procedures for use in cases of otitis media purulenta. The essential procedures are directed toward these desiderata: a clean nose and a reduction of sinus drainage, postnasal drip, adenoidal reaction and tubal catarrh with its poor drainage of the middle ear. This is a logical sequence.

Now, lest some one asks, "How do you suppose ionization works?" let me add that if it is found out I should like to know. The method does not precipitate zinc, nor does it produce any obvious gross changes in the tissue. Sage has analyzed, polarized, sectioned and precipitated material and has found no zinc. It is questionable if solutions of the other heavy metals do not work as well. Some workers believe that the current kills the organisms. Lierle and Sage [1] and Kruger killed bacteria in twenty-four hours with 20 milliamperes, but Zeit concluded that this was due to the products of electrolysis at the poles (oxygen and hypochlorous acid). Zinc sulphate alone may do the work, but this is improbable as many and better bactericides in all sorts of strengths and applied by all sorts of methods have been tried with equally mediocre success. The solution does not permeate all the cellular structure any more than does the fluid used in douching. The question of how ionization acts still stands unanswered.

I am convinced, however, that those who are fatigued with treating patients with persistently discharging ears will find more than occasional satisfactory results in the use of ionization. The method is innocent, and if care is taken there should be no electric shock.

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1. Lierle, D. M., and Sage, R. A.: Underlying Factors in Zinc Ionization, Ann. Otol., Rhin. & Laryng. **41**:359 (June) 1932.

1805 Chestnut Street.

ASSOCIATION OF TOXIC DEAFNESS WITH TOXIC AMBLYOPIA DUE TO TOBACCO AND ALCOHOL

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The purpose of this paper is to present the otologic findings in thirty-six cases of toxic amblyopia due to tobacco and alcohol. During the study of these cases by one of us in the eye service of the Massachusetts Eye and Ear Infirmary the idea occurred that there might also be some associated changes in the ear. This possibility was considered not only because the occurrence of toxic deafness is well known, but because of the functional and anatomic similarity between the spiral ganglion cells of the cochlea and the ganglion cells of the retina. The latter are believed by some observers to be the site of the primary lesion in the eye.

Tobacco-alcohol amblyopia is a definite clinical entity. It is characterized by a centrocecal scotoma, that is, a scotoma extending between, and including, the blindspot and the fixation area. The scotoma is larger for red than for blue. The involvement of the fixation area, which corresponds to the fovea centralis, causes a diminution in visual acuity. The lesion is probably always bilateral and occurs more frequently after the age of 40, but it has been reported in a 13 year old child. Our youngest patient was 25 years of age.

The amblyopia is really of an island type, that is an island of decreased vision in an otherwise normal field. Generally the patient states that he has consumed either tobacco or alcohol, more frequently both. If no other toxic agent can be found to account for the condition, a probable diagnosis of tobacco-alcohol amblyopia is made. If when the patient abstains he has considerable improvement in vision, often a return to normal, a positive diagnosis is justified. The amount of tobacco and alcohol used may be large or small, and the development of pathologic changes probably depends more on an idiosyncrasy to these agents than on the quantity consumed.

In this study, in addition to the ophthalmologic and otologic examinations there were numerous consultations at the Massachusetts General Hospital. General medical and dental examinations and examinations of the nose and throat and the genito-urinary tract were made. Wasser-

From the Massachusetts Eye and Ear Infirmary, Boston.

mann tests of the blood were made on most of the patients and observations on the spinal fluid were made on a small series. Painstaking check-up and follow-up were instituted by the ophthalmologic service. A complete analysis of a larger series of cases observed in the ophthalmologic service during the past two years will be published in a subsequent communication. At this time we wish only to point out an interesting association of tobacco-alcohol amblyopia with toxic deafness.

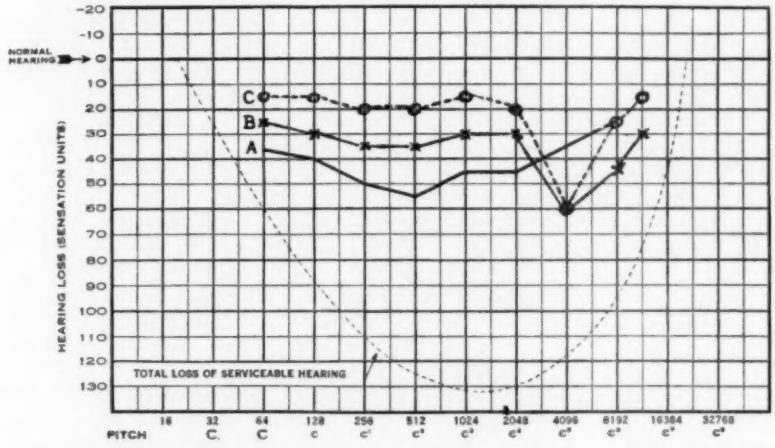
Thirty-six patients were referred to the otologic service for examination. A number of these had, unfortunately for the purpose of this report, shown a great deal of visual improvement when they were first observed in the otologic clinic. There was only one woman in the group, and all the patients belonged to the white race. Italian-born patients predominated. Occupations had no significance, and a great number of the patients were listed as unemployed. The usual duration of the ocular symptoms was several months. The average amount of pure alcohol consumed in a week was 28.6 ounces (845.7 cc.), and the average amount of tobacco smoked, 8.9 ounces (254.3 Gm.). Alcohol was most frequently consumed as wine, especially of the home-made variety; tobacco was most frequently used in pipe smoking.

In the clinic for diseases of the ear all the patients had a thorough otologic examination; this included a routine hearing test and audiogram. A relatively small number complained of deafness or had any otologic symptoms.

In order to make sure that our findings could not be attributed to the usual changes associated with increasing age or to mechanical errors in the audiometer a control group was used. The persons in this group were chosen so that the age was approximately the same in the control group as in the toxic group and also because they had no definite otologic symptoms. Twenty-two patients were selected from the wards for patients with ocular diseases, and these patients were examined also from an ophthalmologic standpoint.

The most outstanding result of the examination was the finding of a notch in the audiogram curve occurring at a frequency of 4,096 vibrations per minute (chart). This defect was a definite drop with a return to the previous level and was nearly always accompanied by a loss of the perception for high tones and a shortening of the bone conduction time. This island area was usually bilateral and occurred so frequently that when it was accompanied by these other changes we regarded it as being of some significance. The average loss for spoken voice was only 8 per cent, so that in the majority of cases symptoms were absent although a considerable number of patients did show a reduction of useful hearing. The large number of patients without symptoms was probably due to the changes occurring at a frequency above the conservation range.

The results of the hearing test and the audiogram of a patient with typical toxic amblyopia, an Italian-born man of 60, are shown on the chart. He stated that he consumed 1½ quarts (1,419.42 cc.) of wine and smoked several cigars daily. Examination was made after he had had symptoms over a period of several months and typical field defects had been noted. Vision was 20/70 in the right and 20/200 in the left eye. The patient had no otologic symptoms, and examination of both ears gave negative results. The Wassermann reaction of the blood was also negative, and dental, medical and genito-urinary examinations by consultants showed no foci of infection.



Typical audiogram of a person aged 60, showing an island defect at a frequency of 4,096 vibrations. This defect was found in a large percentage of the patients tested and was not present in the control persons. *A* indicates the audiometer readings for a person with normal hearing tested with the D-80904 bone conduction receiver. *B* indicates the results of a test on the left, and *C*, the results of a test on the right ear of a patient with typical toxic amblyopia. The amount of loss of hearing was 5 per cent on the right and 7.5 per cent on the left side. The percentage of loss of hearing for speech is approximately equal to the average reading at 512, 1,024 and 2,048 vibrations multiplied by 0.8.

The results of auditory tests made in a sound-proof room 20 feet (6.09 meters) in length are shown in the following tabulation.

Left Side	Test	Right Side
20/25	Whisper	15/25
20/35	Conservation	20/35
20,000	Upper limit	20,000
64	Lower limit	64
30/10–	Rinne	26/9
±	Weber	±

The audiogram is shown in the illustration.

The results of the survey showed that twenty-one patients had changes strongly suggestive of island deafness. Of these, thirteen showed a positive bilateral notch at a frequency of 4,096 vibrations; in seven tests one ear only showed the notch, and in one case the drop was at 1,024 vibrations.

The control group included one person with these changes, but they were not specifically typical. Nearly all the patients with amblyopia showed signs of nerve involvement in addition to the island defect. To say that this was entirely due to toxic causes associated with the use of tobacco and alcohol would be erroneous. From the table it can be seen that the percentage of nerve deafness in the control group is much higher than in the toxic group when not associated with the island change. This simply means that a considerable amount of the associated signs of nerve involvement in the patients with island changes may be due to age.

*Results on Tests on Patients with Toxic Amblyopia and on Controls**

	Number of Patients	Percentage	Number of Controls	Percentage
Notched or island deafness	21	58	1	4
Nerve deafness only	4	11	7	32
Middle ear deafness only	2	6	0	0
Mixed deafness only	3	8	1	5
Normal hearing	6	17	13	59
Total	36	100	22	100

* The average age for the patients was 55; for the controls, 53.

The reduction of useful hearing is more marked in the patients with tobacco-alcohol amblyopia. Normal hearing as shown by the ordinary tests is present in 59 per cent of the control group and in only 17 per cent of the toxic group.

Improvement has not been studied fully to date. Only three patients returned for an otologic check-up. Two showed improvement with a disappearance of the notch, and one of these showed an improvement in the results of the whole hearing test. The third had no auditory change although vision had become much better over the same period.

COMMENT

Pathologic reports suggest that the site of the ocular lesion is in the ganglion cell of the retina and in the nerve fibers of the papillomacular bundle.[1] The otologic lesion is probably in the functionally and anatomically similar spiral ganglion cells of the cochlea. Other possibilities may be a toxic lesion of a small area of the organ of Corti or a specific

1. de Schweinitz, G. E.: The Toxic Amblyopias, Their Classification, History, Symptoms, Pathology and Treatment, Philadelphia, Lea Brothers & Co., 1896.

neuritis of the nerve fibers correlated with the perception of these tones. Recent investigations made at the Harvard Medical School by Davis, Lurie and their co-workers lead one to think that a specific lesion of the organ of Corti is not the explanation, the evidence pointing more to the ganglion cell area. The work on stimulation deafness by the same investigators [2] has shown that the involvement of the organ of Corti is constantly over a wider tone area that that observed in the patients of this series.[3] It tends, therefore, to favor a neuritis of the cochlea nerve, more probably direct involvement of the spinal ganglion. Postmortem examination will be the final proof.

SUMMARY

1. Toxic deafness frequently accompanies toxic amblyopia associated with the use of tobacco and alcohol.

2. A large percentage of the patients showed an island defect at a frequency of 4,096 vibrations. This was not observed in the control group and is quite comparable to the island defect found in the visual fields of patients with amblyopia.

3. There is a general reduction in the perception of high tones and a lowering of the bone-conduction time. In comparison with a control group of the same age this is shown to be due in part to toxic causes rather than to the usual senile defects.

4. There is probably an improvement in hearing function accompanying the improvement in vision on abstinence.

2. Lurie, M. H.; Davis, H., and Derbyshire, A. J.: The Electrical Activity of the Cochlea in Certain Pathologic Conditions, Ann. Otol., Rhin. & Laryng. **43**:321 (June) 1934.

3. Lurie, M. H.; Davis, H., and Derbyshire, A. J.: Stimulation Deafness with a Fixed Tone, to be published.

Clinical Notes

EXPERIMENTALLY INCREASED INTRACRANIAL PRESSURE

WILFRED BRODGEN, A.B., URBANA, ILL., FRED A. METTLER, PH.D., AUGUSTA, GA., AND ELMER CULLER, PH.D., URBANA, ILL.

Patients with increased intracranial pressure usually present complications which interfere with an answer to the question as to what part of the syndrome is produced by pressure on nerve cells and what part by cerebral anemia. The present work was designed to find whether a rise in intracranial pressure affects auditory sensitivity.

Two animals were trained to flex the right foreleg whenever a sound stimulus was given. (Pure tones of 125, 1,000 and 8,000 cycles were used as stimuli.) As the tone was made progressively weaker, the animal continued to react until the tone became no longer audible, whereupon the response ceased. In this way it was possible to determine the animal's acuity of hearing. After the animal's normal threshold of hearing had been ascertained, a threaded metal tube was screwed into a trephine opening in the skull, directly beneath which was an incision in the dura. This was done under complete anesthesia, and it allowed the intracranial pressure to be raised directly. On recovery from the narcosis, the dog was brought to the test room, and connections were made from the skull to a saline pressure bottle. A direct manometer in the circuit provided a means of measuring the pressure used.

In the first animal, the acuity of hearing following operation was (no elevation of pressure) 78 decibels[1] (for the tone of 125 cycles), 97 decibels (1,000 cycles) and 74 decibels (8,000 cycles). When the intracranial pressure was raised to 29 cm. of saline solution, the acuity became: 72 decibels (125 cycles), 88 decibels (1,000 cycles) and 62 decibels (8,000 cycles).

In the second animal, in order to avoid the difficulties which proceed from raising the intracranial pressure above the diastolic blood pressure, ephedrine (5 mg. per kilogram) was injected just before the intracranial pressure was raised. Immediately prior to raising the pressure, the animal's acuity for a tone of 1,000 cycles was 88 decibels. When the intracranial pressure was raised to 150 mm. of mercury, the limen for this tone was 80 decibels. The pressure could not be raised above 200 mm. of mercury without producing symptoms of collapse. At 200 mm. of mercury the acuity for 1,000 cycles remained at 80 decibels. On releasing the pressure entirely, the limen returned to 88 decibels.

From the Laboratory of Animal Hearing, University of Illinois, and the University of Georgia School of Medicine.

Communication 22 from the Animal Hearing Laboratory, established and maintained with aid from the Research Trustees of the American Otological Society. Special assistance from the Elizabeth Thompson Science Fund and the American Academy of Arts and Sciences is gratefully acknowledged.

1. The decibel is a unit of attenuation of sound. A sound attenuated to a value of 81 decibels is weaker than the same sound attenuated to 80 decibels by 1 unit of attenuation.

SUMMARY

It seems from these results that auditory acuity is only slightly impaired by an increase of intracranial pressure over a level which would include practically all pathologic processes. It further appears that relatively complex cortical activity (conditioned response) is possible at such levels of pressure, and that the chief effect of increased intracranial pressure is not due to an effect of pressure on nerve tissue, but to vasomotor failure with consequent cerebral anemia. In cases in which there is impairment of auditory acuity with increased intracranial pressure, a definite involvement of the auditory pathways or direct pressure, as from a neoplasm on the nerve, is to be suspected.

A TRACHEOBRONCHIAL ENDOSCOPE

J. MATHEWS ROBISON, M.D., HOUSTON, TEXAS

This instrument is an adaptation of the principle of the nasopharyngoscope to bronchoscopy. This bronchoscope has a slot in the distal end which holds the endoscope in place but which is not deep enough to prevent it being passed beyond the end of the bronchoscope.

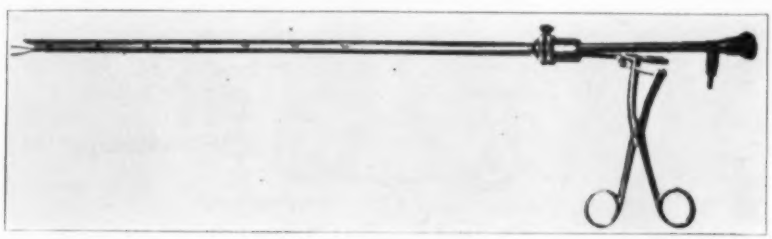


Fig. 1.—The endoscope and the grasping forceps in place in the bronchoscope.

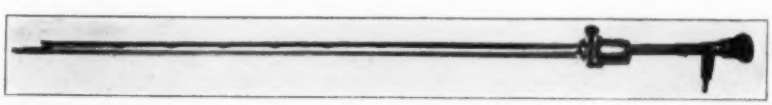


Fig. 2.—The bronchoscope used as a spatula through which the endoscope is passed into regions too small for the bronchoscope to enter.

The endoscope is longer than the bronchoscope, which allows the distal end to be passed into regions too small for the bronschoscope to enter. This excessive length also removes the eyepiece and battery connection from the proximal end, allowing the use of grasping forceps to better advantage.

The endoscope employs the system of lenses used in the nasopharyngoscope, giving an erect, magnified image, except that the terminal lens is so set that it gives a better view forward in its longitudinal axis.

The bronchoscope may be introduced by using the regulation removable light carrier. The light carrier may be removed and the endoscope inserted, or the bronchoscope may be introduced with the endoscope in place.

From the Houston Eye, Ear, Nose and Throat Hospital.

I have used this instrument for about two years and believe that it has a definite field of usefulness in the diagnosis and study of certain types of lesions and foreign bodies in the trachea and bronchi.

The instrument was made for me by the American Cystoscope Makers, Inc., New York.

AN IMPROVED PACKING FOR SEPTUM RESECTIONS PERMITTING NASAL RESPIRATION

SIDNEY H. GIDOLL, M.D., SAN FRANCISCO

Working on the major premise that it is preferable to pack the nasal passages following resection of the septum in order to acquire more perfect approximation of the flaps of mucous membrane and to avoid the formation of a hematoma, the problem presents itself as to the proper type of packing to be employed.

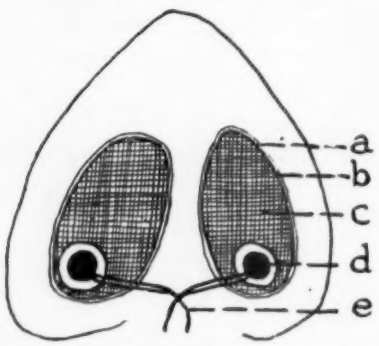


Diagram of an improved method of nasal packing, showing: *a*, the left naris; *b*, the Penrose tube; *c*, the gauze packing within the tube; *d*, the rubber catheter, and *e*, the heavy black silk thread.

The one most commonly used in the past consisted of gauze strips lubricated with petrolatum, but this was found to abrade the delicate mucous membrane of the septum, and removal tore the newly formed adhesions and caused fresh bleeding. To obviate this difficulty, Sewall [1] described a modification of the method of packing with gauze. There was first inserted a piece of gutta-percha tissue which was fitted against the septum and curved outward to protect the upper part, the floor and the turbinates; into this tunnel the gauze tape was tightly packed.

Roth [2] described the objections from the patient's standpoint to a completely obstructed passage. His solution consisted of first inserting a piece of rubber tubing from a catheter into each nasal fossa for about 2 inches (5 cm.), allowing it to rest on the floor of the nasal passage. Gauze strips lubricated with petrolatum were then packed into the nasal chambers above the tubes.

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1. Sewall, E. C., in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1930, p. 75.

2. Roth, Emanuel: A Method of Nasal Packing Following Submucous Resection of the Septum Which Allows Free Nasal Respiration, Arch. Otolaryng. **4**:732 (May) 1931.

The objectionable feature of abrasion from the contact of the gauze and the flaps of the mucous membrane confronts one again in the latter method; so the only ideal method of packing appears to be a combination of the two methods described.

A section of Penrose drainage tubing, ½ inch (1.2 cm.) is tightly packed with gauze and cut into 3 inch (7.6 cm.) lengths; a no. 16 F catheter is inserted along the outer edge of the tubing and is cut off flush with the terminus at the 3 inch mark. A heavy black silk thread is sewed through one end, penetrating the external covering of gutta-percha tissue, the intervening gauze and the rubber tubing within. After being sterilized and when ready for use, two tubes are lubricated with sterile petrolatum, and one is inserted into each nasal fossa, resting on the floor of the nose. The tubes can be cut off as short as necessary prior to insertion, to meet the requirement of the individual nasal fossa.

With an unusually large nasal chamber, more than one gutta-percha tubing can be placed in one nasal fossa. For this purpose I have prepared a second batch of ⅜ inch (0.9 cm.) Penrose tubes, which are also packed tightly with gauze. In this set no rubber catheter tubing is inserted, but the same type of black silk thread is sewed at one end. If, as stated before, the nasal chamber is so roomy as to accommodate more than one Penrose tube on each side, the ½ inch tube is placed on the floor of the nose, and the ⅜ inch tube is placed directly above the larger one.

After insertion of the tubes into each nasal fossa the silk threads on one side are securely tied to those of the other, a small piece of gauze being first placed next to the skin to prevent the latter from being cut. Adhesive strips are then placed across the nares to avoid expulsion of the tubes outward, care being taken to keep patent the lumen of the rubber catheter on each side.

After twenty-four hours, the tubes can be removed en masse merely by exerting traction on the black silk thread.

516 Sutter Street.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

ANATOMY AND PHYSIOLOGY OF THE EAR

PHILIP E. MELTZER, M.D.
AND
CHARLES N. GETTES, M.D.
BOSTON

A review of the current literature in the field of otolaryngology is again presented in abstract form. We have made use of abstracts of articles which have been published by others, and this has facilitated our work immensely. Many articles which should have been reviewed here have been omitted because good abstracts were not available and a lack of time has prevented us from preparing them.

For the first time since this series of reviews was instituted the most fertile investigations on the anatomy and physiology of the ear have been made by Americans. In this country otologists are particularly grateful for the stimulus given to the study of the physiology of hearing by Wever and Bray. To Davis, Lurie, Crowe and Cullen and their co-workers much credit must be given. To Lorente de Nó, Tait and McNally acknowledgment is made of their excellent contributions, particularly on anatomy but also on vestibular physiology. It is earnestly hoped that these investigations will continue and that American otologists will give the workers support and encouragement.

As usual the high standard of research by foreign investigators has been maintained, though reports of their studies have not been as numerous as in previous years.

Excellent articles have appeared on the gross anatomy of the temporal bone which have a practical bearing on the surgical treatment of disease, particularly when the petrous portion is involved.

ANATOMY, HISTOLOGY AND EMBRYOLOGY

Ruskin,[1] in a well illustrated article on the venous circulation of the petrous bone, approaches his subject from two angles and states that the earliest sign of involvement of the petrous bone is a disturbance

1. Ruskin, S. L.: The Venous Circulation of the Petrous Bone and Its Clinical Significance, Ann. Otol., Rhin. & Laryng. **42**:961 (Dec.) 1933.

of circulation in its venous network and that there are two outstanding syndromes: one described by Gradenigo, which is characterized by involvement of the abducens nerve and orbital pain, and another that represents a syndrome which Ruskin here suggests, which is characterized by "edema of the lower lid on the same side, temporomaxillary orbital pain and trismus."

After a careful description of the venous circulation and the pathways of infection to the petrous apex, as described by Schwalbe, Quain, Cunningham, Dorello, MacEwen and Pietrantoni, Ruskin concludes (1) that the venous pathways of the temporal bone play a leading rôle in the dissemination of infection from the tympanic cavity and in the causation of intracranial complications; (2) that early extension of involvement of the venous system can be recognized clinically; (3) that the Gradenigo syndrome should be considered as evidence of venous engorgement of the group of tympanic veins that empty into the inferior petrosal sinus; (4) that the syndrome of temporomaxillary orbital pain, trismus and edema of the lower lid has a significance similar to that of the Gradenigo syndrome but represents venous engorgement of the veins of the tympanic cavity that drain anteriorly into the pterygoid plexus and middle meningeal veins, and finally that the orbital and trigeminal symptoms may be induced by mechanisms affecting the pterygoid and middle meningeal venous systems other than those of petrosal origin, and must be carefully differentiated in order to avoid unwarranted surgical invasion of the petrous bone.

In a well illustrated article dealing with the innervation of the macula sacculi in man, based on serial sections of adult human temporal bones, Hardy [2] presents a description of "small bundles of nerve fibres that pass from the cochlear ganglion, in a region near its basal end, to the posterior tip of the macula sacculi." He states:

> The course of these bundles is through small bony channels about a millimeter in length. The general direction from ganglion to macula is upward and medialward and approximately in the plane of the "vertical" sections. The term cochleosaccular nerve is proposed for this group of nerve bundles. The belief is expressed that this nerve to the sacculus is a true branch of the cochlear nerve, and not merely an anastomotic twig from the vestibular nerve.
>
> The saccular nerve from the superior vestibular division described by Voit for mammals is of constant occurrence in the adult human ear. Oort's vestibulocochlear anastomosis is identified in some but not in all series observed. The general course and location relative to the labyrinth as a whole is described for these two nerve branches, as well as for the cochleo-saccular nerve.

Hardy contends that the "macula sacculi in man therefore is innervated by branches from all three divisions of the eighth nerve. The

2. Hardy, M.: Observations on the Innervation of the Macula Sacculi in Man, Anat. Rec. **59**:403 (July 25) 1934.

ramus saccularis of the inferior division of the vestibular nerve is much larger than either Voit's nerve or the cochleo-saccular nerve; the latter is the smallest of the three."

Almour,[3] after a careful study of 24 well pneumatized temporal bones, calls attention to a distinct tract extending from the middle ear to the tip of the mastoid. He says: "Starting at the mastoid tip, this tract is found to follow from the tip cell upward and inward, along the posterior canal wall, and then directly inward, overlying the lateral sinus, until it reaches an area located posterior to the jugular bulb. This tract then runs underneath the facial nerve to emerge in the middle ear at the innermost part of the junction between the posterior and inferior tympanic walls." This he clearly demonstrated by the use of dyes and by means of dissection with wire probes in place.

Almour feels that the presence of this tract explains tenderness of the tip of the mastoid coincident with acute otitis media and mastoiditis, when this is the only site that is tender to touch throughout the course of the disease.

Henry,[4] in a commendable study of the lymphatic vessels and the movement of lymph in the ear of the rabbit, presents the following summary:

1. The rabbit's ear possesses a rich lymphatic supply of both superficial and deep vessels, with 8-12 collecting vessels, which follow the course of the main blood vessels.

2. The vessels of the inner side of the ear pass through the cartilage to join those of the outer side.

3. The total area of the lymphatics of the ear, as shown by surface projection, is approximately equal to that of the blood vessels.

4. By the introduction of cannulae into lymphatic vessels draining the ear, it was possible to secure data regarding the amount of lymph flow. This was found to be variable, but always extremely small, even when massage was freely used. Calculations indicated figures ranging from 0 to 0.0125 cu. mm. per square millimeter of lymphatic surface per hour spontaneous flow, and to a maximum of 0.084 cu. mm. per square millimeter of lymphatic surface per hour after massage.

5. Studies of injected granules indicate that there was some obstruction of lymph flow in the thinner but none in the thicker parts of the older type of "preformed tissue" chamber.

6. By injected granules it was possible to prove definitely that the suspected openings between lymphatics and the supernatant fluid seen following the installation of the "preformed tissue" chambers are real openings into lymphatics, and that they may persist for as long as 13 days.

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3. Almour, R.: The Evolution of the Mastoid Tip Cell as a Cell System Separate from the Remainder of the Mastoid Cells, and Its Significance (Preliminary Report), Laryngoscope **43**:797 (Oct.) 1933.

4. Henry, C. G.: Studies on the Lymphatic Vessels and on the Movement of Lymph in the Ear of the Rabbit, Anat. Rec. **57**:263 (Oct. 25) 1933.

Myerson [5] and his co-workers studied the petrous portions of 200 temporal bones obtained from the skulls of persons ranging in age from 7 months of fetal life to 89 years.

Pneumatization of the petrous apex was encountered in 11 per cent of the specimens. Pneumatization does not appear to occur as frequently as has been supposed, especially in children. In 36 specimens from children up to and including 7½ years there were no pneumatized apexes.

The arrangements of cells of the pneumatized bodies were recorded, and a simplified grouping of the cell channels is offered.

The cellular structure of the mastoid portion and that of the petrous is not always the same. Thirty-eight per cent of the mastoid processes but only 11 per cent of the petrous portions were pneumatized. Twenty-five per cent of the mastoid processes were diploic as compared with 11 per cent of the petrous pyramids.

The best developed cell channels were the jugular bulb group (the subcochlear and the posterior cochlear) and the anterior surface cells above the cochlea.

In 4 of the 100 skulls the petrous apex was pneumatized on one side and not on the other.

In this series there was a greater incidence of pneumatization in the skulls of colored than in those of white persons.

Lurie,[6] in an excellently illustrated paper on the evolution of the cochlea, describes the development from the lowest to the highest vertebrates. He presents definite evidence of the way this organ may respond to sound. Beginning with the phylogenic importance of the organ of hearing in the fish, the development of the organ of hearing in the amphibian, reptile and bird and the relationship of the organ in the lowest to that in the highest forms of mammals, he concludes that the mammalian cochlea is:

(1) A development from two organs for hearing of which the lagena is the more primitive.

(2) That with the cochlea taking over the function of the lagena it should respond in a similar manner as the lagena does.

(3) The mammalian cochlea may therefore be capable of responding to sound in two ways: (*a*) stimulated as a whole in noise or low frequency sounds; (*b*) stimulated in specific regions for higher frequencies and musical tones.

Altmann [7] reports the postmortem findings of an island cartilage in the upper wall of the internal acoustic meatus in a child aged 2 days with microtia and congenital atresia of the external auditory meatus

5. Myerson, M. C.; Rubin, H., and Gilbert, J. G.: Anatomic Studies of the Petrous Portion of the Temporal Bone, Arch. Otolaryng. **20**:195 (Aug.) 1934.

6. Lurie, M. H.: Phylogenic Development of the Cochlea, Tr. Am. Otol. Soc. **23**:210, 1933.

7. Altmann, F.: So-Called Persistent Cartilaginous Foci in the Region of the Bony Labyrinthine Capsule, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:297 (Sept. 21) 1932; abstr., Arch. Otolaryng. **17**:586 (April) 1933.

on the left side. According to Bast, five ossification centers are involved in the development of the bony internal acoustic meatus, all of which are fused in the 19½ week embryo. Normally there are no cartilage rests in the superior wall of the acoustic meatus after this period. However, Altmann thinks it possible to attribute the island of cartilage to a cartilage rest, which is usually present only a short time and rapidly disappears. Analogously, in the course of endochondral ossification of the long bones, islands of cartilage are sometimes excluded during the fetal period or later by injurious influences and may persist while the remainder of the cartilage ossifies. They constitute a simple chorista or may proliferate and give rise to tumors.

Wünn [8] studied the cupola in the intact ampullae of the semicircular canals in the labyrinth of the pike according to Steinhausen's method, immediately after killing the fish. By the term cupola, the author means only the structure rising above the sensory epithelium of the crista in the ampulla of the semicircular canals, in contrast to Werner and Wittmaack, who designate the membrane extending from the otoliths to the macula in the utriculus and sacculus as the cupola. The author's studies confirm and amplify the observations made by Steinhausen. The vitally fresh cupola has a complicated morphology and is much larger than was formerly assumed. In the fixation of the cupola, even with the mildest fluids, severe shrinking of the tissue results in a marked change of its size and shape. It may therefore be assumed that the finer histologic structure of the fixed cupola may in no way be compared with that of the fresh unfixed cupola.

Anson,[9] in another histologic study of the endolymphatic appendage, after alluding to the work reported by others, especially Guild, presents the following summation:

> The entire endolymphatic appendage is, in its early development, at first a digitiform and then a lanceolate diverticular process of the otocyst. Very soon after the whole appendage becomes differentiated into the commonly recognized subdivisions, ductus and saccus, a third segment appears, in the form of a narrow, distally placed, prolongation. This third subdivision, the distal projection, is unmistakably present in the embryo of 19 mm., and seems to owe its formation not only to elongation of the terminal third and the primitive appendage, but also to accompanying construction of this part. Then, while retaining its typical shape, further extension occurs through the 27-mm. stage—the length of the entire endolymphatic appendage being tripled or quadrupled while its greatest width is but doubled. Thereafter, as shown by the most advanced stages included in our reconstructions, the distal projection retains its long thin form, while the saccus becomes

8. Wünn, F.: Cupola Terminalis in Labyrinth of Pike, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia Oto-laryng.) **22**:481 (June) 1932; abstr., Arch. Otolaryng. **17**:588 (April) 1933.

9. Anson, B. J.: The Distal Projection of the Endolymphatic Sac in Human Embryos, Anat. Rec. **57**:53 (Aug. 25) 1933.

more bulbous; the projection is still present in the most advanced series available, the 74-mm. embryo. Concurrently, with the alterations described above, the ductus becomes, in general, progressively more elongate. . . .

Histologically, the wall of the distal projection, like that of the saccus and of the ductus, consists of a single layer of cuboidal epithelial cells. In the region where the sac definitely narrows to become the dorsal extension, it is approximately ten cells in circumference; the number of cells gradually decreases to four or five in the terminal fourth, and the lumen becomes progressively smaller; the latter usually disappears in the distal reaches, so that the tip of the process is a solid cord, composed, in transverse section, of four, three or even fewer cells.

The distal prolongation of the endolymphatic sac is, as far as our observations indicate, restricted in its occurrence to the embryo of man; it was not seen in the embryos of the other ten mammalian species included in this study.

Although the author is unable to determine the fate of this striking prolongation of the saccus endolymphaticus in the fetal or the adult ear, he suggests that "the structure is perhaps represented by the very irregular, or occasionally even contorted, termination of the endolymphatic duct observed in serial section of ears from some postnatal specimens."

Bast,[10] in a comprehensive study of the origin, development and significance of the fissula ante fenestram, "an irregular projection of the periotic connective tissue extending from the angle of the junction of the vestibule and the scala vestibuli, through the otic bony capsule to the periosteum of the middle ear just below the pulley of the tensor tympani muscle," states the following conclusions:

The tissue of the fissula arises, as does the periotic tissue, from precartilage. 1. The precartilage undergoes a regressive change to form a vacuolated reticular tissue. 2. With the entrance of blood vessels from the middle ear, the tissue undergoes new growth, filling the fissula with fibroblasts which form a fine fibrillar connective tissue. 3. At the periphery of the fissula this connective tissue changes to cartilage, whereas in the center of the fissula a loose vacuolated vascular connective tissue is formed. . . .

In most cases the tissue of the stapes ligament is continuous with the tissue of the fissula. The connective tissue cells of the annular ligament are arranged in a radial direction, or the long axes of the fibroblasts lie at right angles to and stretch across the stapes joint. These cells retain this same direction as they continue into the fissula, which communicates at a right angle with the stapes joint. In a small percentage of cases the communication with the stapes joint does not occur. . . .

No attempt is made in this paper to explain this communication of the fissula with the stapes joint; it is intended only to present this relationship as an anatomic fact. However, it may be well to bear this relationshp in mind in studying otosclerosis, since it usually occurs in this region and often causes anklyosis of the stapes.

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10. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1 (July) 1933.

Oesterle [11] states that "interglobular spaces" or remains of primitive cartilage are characteristic of the labyrinthine capsule. They do not occur in bone anywhere else, with the exception of the auditory ossicles, which resemble the bone of the labyrinthine capsule in this respect. The most likely explanation is that the labyrinthine capsule and the ossicles reach their full development before birth; therefore, there is a persistence of certain embryonic features which in long bones have become lost in the course of further development.

These special features in the histology of the ossicles were studied by Oesterle in serial sections of 30 temporal bones of all ages, and numerous sections and diagrams appear in the text. He found that the arrangement of the fibrous element of the bone is also fundamentally different from that found in ordinary long bones. In the labyrinthine capsule and auditory ossicles the fibrils are not arranged in the form of lamellae but form comparatively large bundles which are interwoven with each other in irregular whorls. Special staining methods are necessary for the study of these structures, and the differences between them and the ordinary lamellar bone are best studied with the binocular microscope. In the irregular whorls (*Strähnenknochen*) a horizontal section presents practically the same appearance as a vertical section.

Steinberg [12] studied serial sections of 30 specimens of temporal bones cut horizontally and of 10 specimens cut vertically to determine whether the "interglobular spaces" favor certain parts of the labyrinthine capsule, as maintained by Mayer and others, and also whether these cartilaginous remains are grouped according to any kind of regular plan.

Manasse first described the interglobular spaces as characteristic of the labyrinthine capsule, and there is general agreement that the presence of these embryonic remains is due to the fact that the capsule completes its development during fetal life.

Steinberg agrees, on the one hand, that certain areas of the capsule are especially rich in interglobular spaces, e. g., near the ampullae, at the tip of the cochlea and at the level of the round and oval windows. On the other hand, she did not find that there is any kind of regular arrangement. In some specimens the interglobular spaces appeared to be arranged in layers parallel to the surface of the labyrinth (Mayer), while in others the arrangement was irregular.

Meyer [13] states that the normal anatomy of the otic capsule is of interest because of the basic difference of the osseous structure of this

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11. Oesterle, F.: The Microscopic Structure of the Ossicles and Their Development, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:311, 1933.

12. Steinberg, L.: The Distribution and Situation of the Cartilaginous "Interglobular Spaces" in the Human Labyrinthine Capsule, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:187, 1933.

13. Meyer, M.: The Normal Anatomy of the Otic Capsule, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:3 (May 6) 1933; abstr., Arch. Otolaryng. **18**:536 (Oct.) 1933.

part of the skeleton and of its mode of development from those of any other bone in the body, and because only an exact knowledge of the normal structure permits recognition of pathologic deviations from it. After describing in detail the normal anatomic structure of the otic capsule in man and its development, he discusses cartilaginous rests, fissures and the ossicles. He utilized monkeys and dogs in his studies because the structure of the capsule in these animals is analogous to that of human beings.

Kelemen,[14] in a study of 14 temporal bones in serial sections cut in the horizontal plane, noted that nontraumatic fissures occurred in the bony capsule of the inner ear. Of these there were two groups: (1) fissures around the lumen of the semicircular canals and (2) fissures near the round window. The specimens were obtained following accidental death, usually due to fracture of the base of the skull. Nevertheless, the author is satisfied that the fissures which he describes had nothing to do with antemorten trauma.

The most commonly found fissure was a split in the bony capsule connecting the recess of the round window and the ampulla of the sagittal canal. It was seen in 5 of 14 specimens. By a careful analysis of the microscopic appearances the author seems to prove that this fissure is not the result of trauma; neither is it an artefact or simply a bony canal due to the presence of a blood vessel running horizontally at that level. He expresses the belief (as does Mayer) that these fissures are caused by some strain, possibly connected with intracranial pressure, which acts on the labyrinthine capsule during life.

In a detailed study of the eighth nerve based on the problem of whether or not there are present in the pathways of hearing individual fibers for the different regions of the cochlea, Lorente de Nó[15a] offers the following conclusions:

1. The fibers from the labyrinth end-organs (cristae and maculae) have been followed from the labyrinth, through the ganglion of Scarpa, into the medulla oblongata.

2. The central division of the vestibular nerve may be divided into five groups of fibers, each having a special distribution.

3. Groups I and II contain fibers from the semicircular canals; group IV, fibers from the utricular macula, and group V, fibers from the saccular macula; it has not been determined whether group III contains fibers from the central part of the cristae or from a region of the utricular macula. Both alternatives are possible because all these fibers are thick.

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14. Kelemen, G.: Fissures of the Bony Portion of the Internal Ear, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:36, 1933.

15. Lorente de Nó, R.: (*a*) Anatomy of the Eighth Nerve: I. The Central Projection of the Nerve Endings of the Internal Ear, Laryngoscope **43**:1 (Jan.) 1933; (*b*) Anatomy of the Eighth Nerve: III. General Plan of Structure of the Primary Cochlear Nuclei, ibid. **43**:327 (April) 1933.

4. The semicircular canals and the maculae have partly different and partly common central representation. The fibers from both maculae have extremely similar distribution.

5. The entire static labyrinth must be considered as a physiological unit; the saccular maculae must, of course, have a function similar to that of the rest of the labyrinth; an acoustic function for it cannot be accepted, because it has connections with the centers and pathways involved in the regulation of equilibrium, but no connection with the pathways and centers involved in hearing.

The conclusions presented by Lorente de Nó [15b] in a further study of the general plan of structure of the primary cochlear nuclei, which he accomplished by selective methods of staining, are as follows:

1. Each cochlear fibre gives branches to no less than thirteen regions of specific structure, where more than fifty different types of cells have been found. Each cochlear fiber establishes connections with hundreds of cells. Endogenous association fibres connect reciprocally the different parts of the primary nuclei.

2. The primary nuclei cannot be considered as "dead beat" synapses, but rather as "selective" synapses, which transmit impulses according to: (a) the afferent impulses; (b) the impulses set up in the nuclei themselves by the cells with short axis-cylinders; (c) the impulses arriving from the upper nuclei via the centrifugal fibres.

Werner [16] studied the effect of changes in air pressure on the labyrinths of guinea-pigs. He states that increases or decreases of air pressure effect regular changes in the height of the epithelium and in the cellular picture of the macula sacculi. These changes are not explainable mechanically but depend on the reactions of the living cells. They are lacking in the macula utriculi. The changes in the macula sacculi, therefore, evidently depend on a specific sensitivity and are perhaps an expression of a functional relation of this macula to atmospheric pressure. This accords with the fact that as yet no other function has been demonstrated for the macula sacculi in mammals.

Albrecht and Schwarz [17] made an extensive study of the histologic structure of the mucous membrane of the middle ear of new-born infants and of fetuses. They believe that there are great individual differences in the structure of the epithelial cells and of the underlying layer of fibrous tissue which can be explained only as inborn variations which are characteristic for each person (constitution).

Wittmaack has questioned Albrecht's researches on the ground that the individual differences in the microscopic structure, particularly of the fibrous tissue cells, are simply postmortem changes comparable to those which set in so rapidly in the organ of Corti after death.

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16. Werner, C. F.: Cellular Reactions in the Labyrinth to Changes of Air Pressure, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:544 (March 20) 1933; abstr., Arch. Otolaryng. **18**:673 (Nov.) 1933.

17. Albrecht, W., and Schwarz, M.: Constitution and Pneumatization, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:50, 1933.

Albrecht and Schwarz believe that the sensulae (*Sinnesendstellen*) of the inner ear are known to undergo rapid postmortem changes but that this factor does not apply to the cellular elements of fibrous tissue.

The fundamental differences of opinion naturally extend into the domain of pneumatization. Albrecht finds no difficulty in explaining all the different types of mastoid processes on a basis of the constitutional variations in the mucous membrane, while Wittmaack's pneumatization theories have as a basis the inflammatory changes which occur in early infancy (hyperplastic otitis).

Wittmaack [18] disagrees with Schwarz's interpretation of the individual morphologic differences observed in the mucosa of fetuses and of the new-born. The latter regards these as signs of genotypic differences in the nature of the mucosa. Wittmaack has seen the differences described by Schwarz but attributes them to postmortem changes of varying intensity and possibly of somewhat varying nature. He thinks that in the mucosa of the middle ear, as in all tissues, the individual genotypic anlage is not morphologically distinguishable and can be recognized only in the different forms of reaction to the various environmental or "phenotypic" influences. When morphologic differences are seen, phenotypic influences have already been at play. All changes in the morphology of the mucosa, except slight ones which may be due to individually different influences of the eustachian tube or of the nasopharynx, are due to early inflammatory processes. Irritants which affect chiefly the epithelial layer provoke chiefly hyperplastic reactions of the mucosa; those which affect chiefly the subepithelial layers determine mainly hypoplastic changes. Either of these two opposing phenotypic factors, or possibly both, may determine the development of the mucosa. Because of the genotypically determined individual susceptibility of the mucosa, the intensity of the changes produced in different persons by inflammatory irritants of the same degree is variable. The intensity of the change may vary in different parts of the middle ear. The stage in the development of the mucosa and in the process of pneumatization in which the phenotypic factors come into play is variable. The interaction of all these factors explains the wide variety of pneumatization types which are observed.

Smith,[19] after reviewing the literature on pneumatization of the mastoid cells as laid down by Alexander, Brock, Cheatle, Logan, Turner, Wittmaack and others, offers in a beautifully illustrated article the following conclusions:

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18. Wittmaack, K.: Nature of Mucosa and Pneumatization, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:261 (Aug. 23) 1932; abstr., Arch. Otolaryng. **17**:585 (April) 1933.

19. Smith, A. B.: The Development of the Mastoid Air Cells, J. Laryng. & Otol. **48**:225 (April) 1933.

The mastoid air cells are formed by (1) resorption of the bony walls of the mastoid antrum by osteoclasts, (2) penetration of the sub-epithelial connective tissue into the spaces hollowed out by these multi-nucleated cells, (3) replacement of the bone marrow by this tissue, (4) degeneration and absorption of the central part of the connective tissue followed by its condensation as a thin layer on the surface of the bone, and (5) proliferation of the epithelium which follows the regression of the connective tissue and remains in contact with it.

The maxillary air cavity develops in a similar manner but the relative absence of bone marrow accounts for its smooth walls compared to the irregular air cell development of the mastoid process.

It is doubtful if the hyperplastic changes in the epithelial layer play any part in preventing development of the mastoid air cells.

The sub-epithelial connective tissue is most abundant when the mastoid air cells are still in the process of development. Once these are fully developed this connective tissue becomes indistinguishable from adult fibrous tissue and forms a very thin layer on the bony wall of the air cell.

The constitution of the osseous tissue of the mastoid process is the determining factor in the development or non-development of the air cells.

Meltzer [20] studied the arrangement of the mastoid cells in relation to the lateral sinus. His conclusions are based on a study extending over a period of twelve years in which dissections of many hundred temporal bones were made. His observations were corroborated in a practical way during several hundred mastoidectomies, in which the exenteration of cells followed according to the position of the lateral sinus. He feels that the position of the sinus determines the arrangement and distribution of cells when they develop. The type of pneumatization is discussed, but Meltzer is emphatic in stating that the arrangement of cells has nothing to do with the type of cell. It does not matter whether the cells are pneumatic, diploic, etc., because the distribution and arrangement depend on the relationship of the lateral sinus plate to the cortex of the mastoid, the digastric crest and other structures. To understand these points fully it is necessary to read the article and see the plate illustrating the various parts and types of mastoid. The author feels that once the course and position of the lateral sinus are known, the distribution of cells is evident, and that, therefore, in the performance of mastoidectomy the operator should leave no hidden groups of cells uncovered or unextenterated.

PHYSIOLOGY OF HEARING

We consider that this is an excellent time to give a résumé of the work done at the Harvard Medical School in the department of otology and otolaryngology under the supervision of Davis and his associates,

20. Meltzer, P. E.: The Mastoid Cells; Their Arrangement in Relation to the Sigmoid Portion of the Transverse Sinus, Arch. Otolaryng. **19**:326 (March) 1934.

Lurie, Derbyshire and Saul.[21] The work has progressed without interruption and has yielded important as well as interesting results. It has centered around the electrical phenomena of the ear itself and of the auditory nerve, but, in addition to the physiologic analysis of these phenomena, much information has been gained as to the exact nature of the physical mechanisms of the inner ear and the interpretation of certain phenomena of hearing.

These investigators have come to the conclusion that the electrical activity at the round window represents simply the response of the cochlea, specifically the organ of Corti. They have recently found that the response of the auditory nerve also enters into this phenomenon. This has served to explain numerous irregularities which were observed in their earlier experiments and has made possible a much better evaluation of the degree to which higher harmonics are introduced by the ear in response to a pure, stimulating tone.

A technic has been perfected by these workers for obtaining "audiograms" from cats and guinea-pigs by electrical methods. They have studied separately and have compared the acuity revealed by the cochlear response and that shown by the nerve impulses in the auditory nerve, and have established norms for experimental animals; these have proved to correspond closely to the curves for human audibility as determined with the same stimulating apparatus. The norms also agree quantitatively with the best data obtained by other workers on animals by means of the study of conditioned reflexes. With this tool Davis and his associates are studying with care the great variety of pathologic conditions of the ear which appear in their experimental animals including acute and chronic infections of the middle ear, both complete and partial congenital absence of the organ of Corti, congenital partial deficiency of the auditory nerve and infection and congestion of the inner ear.

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21. (*a*) Saul, L. J., and Davis, H.: Action Currents in the Central Nervous System; Action Currents of the Auditory Tracts, Arch. Neurol. & Psychiat. **28**:1104 (Nov.) 1932. (*b*) Davis, H.; Derbyshire, A. J., and Saul, L. J.: Further Analysis of the Electrical Phenomena of the Auditory Mechanism, Am. J. Physiol. **105**:27 (July) 1933. (*c*) Davis, H., and Saul, L. J.: Auditory Action Currents, Am. J. Psychol. **45**:358 (April) 1933. (*d*) Davis, H.; Derbyshire, A. J.; Lurie, M. H., and Saul, L. J.: Further Analysis of Cochlear Activity and Auditory Action Currents (with Demonstration of Audible Response), Tr. Am. Otol. Soc. **23**:106, 1933. (*e*) Lurie, M. H.: Phylogenic Development of the Cochlea, Ann. Otol., Rhin. & Laryng. **42**:1069 (Dec.) 1933. (*f*) Davis, H.; Derbyshire, A. J.; Lurie, M. H., and Saul, L. J.: The Electric Response of the Cochlea, Am. J. Physiol. **107**:311 (Feb.) 1934. (*g*) Lurie, M. H.: Recent Experimental Work on the Physiology of Hearing: Its Significance to the Otologist, Laryngoscope **44**:488 (June) 1934. (*h*) Lurie, M. H.; Davis, H., and Derbyshire, A. J.: The Electrical Activity of the Cochlea in Certain Pathologic Conditions, Ann. Otol., Rhin. & Laryng. **43**:321 (June) 1934. (*i*) Davis, H.; Derbyshire, A. J., and Lurie, M. H.: A Modification of the Auditory Theory, Arch. Otolaryng. **20**:390 (Sept.) 1934.

The phenomenon of making tones has been analyzed experimentally, and it has been shown that it depends largely, if not entirely, on the nature of the conduction of nerve impulses. Masking does not occur in the cochlear response; the two simultaneous tones, or a tone and a noise, simply add together. In the nerve fibers, however, there is competition instead of addition, owing to the necessity for a period of recovery in each fiber following the transmission of an impulse. This relationship had been suggested theoretically by previous workers, but Davis and his colleagues have demonstrated it directly. The masking is peripheral, not central; the impulses aroused by pure tones and those aroused by noises travel through the same nerve fibers. There is no justification for assuming any separate mechanism for the reception of noise.

The impulses in the auditory nerve have been studied with respect to the delay following the arrival of sound waves at the ear, their magnitude in relation to the intensity of stimulation, and the effects of fatigue which they show, and these investigators have arrived at three general conclusions:

1. The fibers of the auditory nerve are precisely similar with regard to rate of conduction, rate of recovery, etc., to fibers of the same size elsewhere in the body.

2. The intensity of a tone is represented by the number of nerve fibers activated and not by the number of impulses per second transmitted to the brain.

3. Effects of fatigue are present in the nerve but do not impose any significant limitation on the power of hearing.

Their investigations of the mechanics of the cochlea have demonstrated the following facts:

1. The cochlear response is developed by the hair cells of the organ of Corti as an electrical potential across the basilar membrane.

2. Stimulation of the nerve fibers is associated with outward movement of the tympanum. No stimulation occurs during the cycle of inward movement.

3. The basilar membrane is tuned like a resonant mechanism with the region which responds to high tones located near the round window and that which responds to low tones at the apex. This conclusion rests on observations made on experimental animals and in cases of stimulation deafness, as well as on a confirmation and extension of the recent experiments of Hallpike and Smith.

4. Evidence has been obtained that activity corresponding to high tones is carried on in a different section of the auditory nerve from that which is activated by low tones.

Davis and his associates are continuing their study of stimulation deafness, and we are privileged to report a résumé of their findings which will be published in full in April 1935. Definite deficiencies of hearing associated with definite pathologic lesions have been produced by exposing guinea-pigs to loud tones for long periods of time. A tone of 600 cycles per second is ineffective in producing such damage, but a tone of 2,500 cycles per second has caused it regularly. The loss of hearing does not correspond to the frequency of the tone but affects rather the range of frequencies to which the ear is normally most sensitive. The lesion associated with this type of deafness is a degeneration of the external hair cells, extending over a rather wide zone and centering in the upper part of the basal whorl and the second whorl of the guinea-pig. In these experiments Dr. E. H. Kemp of Clark University and Dr. Morgan Upton of the Department of Biology at Harvard University collaborated.

All of their results constitute strong confirmation, to the point where doubt is scarcely permissible, of a resonance theory of hearing along the lines originally proposed by Helmholtz but with the modification that the localization of resonance to a particular tone is not sharp but is distributed over a fairly extensive region of the basilar membrane. It is believed that it applies to low as well as high tones, although the evidence is more complete for the high tones.

Regarding the auditory centers in the brain, material is being prepared for future publication. Davis and his colleagues are beginning a direct study of the auditory regions of the cerebral cortex, and have devised suitable apparatus for registering the relatively slow electrical fluctuations of the gray matter. The pattern of the activity is characteristically different from that of the visual, motor and other regions of the cortex and offers a promising tool for analysis of the central mechanisms of hearing. Stimulation by sudden noise causes an abrupt electrical response superimposed on this activity, and continued stimulation may cause a pronounced modification of the pattern. The frequency of a stimulating tone is never reproduced, although the rhythm of successive clicks may be recognized up to 50 or 60 per second. The investigation by means of this type of experimentation is just being started, and great hopes of its future development are entertained.

At Johns Hopkins University Hughson and Crowe [22] have been carrying out a detailed experimental investigation of the physiology of the ear and offer the following conclusions:

1. Using the Wever and Bray phenomenon and with an accurate method of measurement of the intensity of tones transmitted through the cat's ear, circum-

22. Hughson, W., and Crowe, S. J.: Experimental Investigation of the Physiology of the Ear, Acta oto-laryng. **18**:291, 1933.

cision of the tympanic membrane has practically no effect on the intensity of tones received through the amplifying apparatus. This confirms in every particular the observation which had been previously reported.

2. Fixation of the tensor typmpani muscle produces a loss of conduction of low tones. It has also been found that the muscle will contract reflexly following stimulation of the pinna, the external auditory canal or the facial nerve.

3. The effect of immobilization of the secondary tympanic membrane with a pledget of cotton previously reported has been repeatedly confirmed. In addition tissue grafts of periosteum placed in the round window niche with sterile precautions grow in that position and when the animals are tested at intervals of from two days to seven weeks transmission upon the operated side is increased from ten to fifty decibels for the different oscillator tones. No other explanation of this phenomenon than immobilization of the secondary tympanic membrane can reasonably be considered at the present time.

4. An accurate and reliable method has been developed for measuring intralabyrinthine pressures and for demonstrating the relationship between cerebrospinal fluid and intralabyrinthine pressures. Using this method it has been shown experimentally for the first time that marked decrease in intralabyrinthine pressure causes a reduction in the intensity of all tones transmitted and usually a complete loss of high tones. Increased pressure has little, if any, effect upon conduction; possibly with extreme increases a slight improvement may result. There is a definite lag in the change produced upon intralabyrinthine pressure, the extremes here not being reached until some time after the limit of change of the cerebrospinal fluid pressure has occurred.

5. A method has been developed whereby bone conduction may be studied experimentally and the intensity of this type of tone transmission can either be improved or made less intense by a variety of procedures.

Guttman and Barrera,[23] after briefly reviewing the work of Wever and Bray, Adrian, Davis, Saul, Derbyshire, Lurie and others and Guttman's own work, report their observations made after sectioning the eighth nerve intracranially in a series of cats. They found that after various periods of survival there was complete degeneration of the cochlear ganglions with the nerve elements and efferent nerve fibers. In each case the cochlear response, when led off from the cochlea, was found to be unchanged. They conclude:

> Since the cochlear response persisted even after the complete degeneration of the nerve elements in the cochlear ganglia, it would seem that nervous elements in the cochlea, at least in the ganglia, play little if any rôle in the production of the cochlear response. The experiments reported in this paper throw no light on the question as to the living tissue or nonliving structure origin of the response.

Gray [24] abstracts three articles by George von Békésy on the physiology of hearing which appeared in the German and French literature

23. Guttman, J., and Barrera, S. E.: Persistence of Cochlear Electrical Disturbance on Auditory Stimulation in the Presence of Cochlear Ganglion Degeneration, Am. J. Physiol. **109**:704 (Oct.) 1934.

24. Gray, A. A.: Review on Some Recent Papers on Hearing, J. Laryng. & Otol. **49**:344 (May) 1934.

and says that Békésy approaches the subject of the theory of hearing by the investigation of the sense of direction of sound as perceived simultaneously by the two ears. By means of a complicated piece of apparatus devised by Békésy sounds were conveyed to both ears simultaneously in such a way that variations in loudness, quality and phase could be changed. It was found that variations in these factors conveyed to the listener the impression that the direction from which the sounds came had varied accordingly.

Békésy attempts to establish an analogy between the sound picture created in the mind by the sense of hearing and the visual image created by the sense of sight. If the sound is heard equally loudly in the two ears, the subject is aware of the fact that it comes from either directly in front or directly behind and can by voluntary effort convince himself as to which is correct. Békésy considers this phenomenon to be analogous to that in vision when the subject looks at a drawing of a truncated cone. By a mental effort he can see the cone as though its truncated end were either nearer to or farther from him than its base, as he chooses. This analogy, however, appears to Gray to be fanciful.

Békésy next relates some experiments on the effects of a momentary sound, such as a report or sudden crack, on the chain of ossicles and the basilar membrane. The sound he employed was that produced by the contraction of the tensor palati muscle without at the same time performing the act of swallowing. The movements of the tympanic membrane were reproduced by means of an oscillograph. He claims to have ascertained the proper tone of the tympanic membrane and says that his results are in close approximation to those obtained by Franke, who experimented on the tympanic membrane in the cadaver. He also experimented with the effect of sound on the basilar membrane, his object being to prove that the sensation of sound is produced by progressive waves traversing the basilar membrane and not by stationary waves. Gray says, however, that "the conditions under which experiments can be carried out are so widely different from natural conditions, that the results obtained cannot be regarded as at all satisfactory."

In a third paper Békésy advances mathematical equations in an attempt to determine the movements of the basilar membrane in response to sound vibrations. In his equation the length of the membrane and the density and coefficient of friction of the fluid are stated, but he ignores the factor of tension by the ligamentum spirale, which, as Gray remarks, "happens to be the most important of all."

None of the three articles is at all convincing to the reviewer, Albert Gray.

Culler, Finch and Girden [25] studied the effects of plugging the round window and disagree with Hughson and Crowe, who claimed that when

25. Culler, E.; Finch, G., and Girden, E.: Function of the Round Window, Science **78**:269 (Sept. 22) 1933.

the round window of the cochlea is made rigid by pressing on it with a plug of moist cotton the perception of practically all tones is increased about 50 per cent. Culler and his associates found that plugging the round window reduced the animal's ability to hear and also the electrical impulses conveyed by the auditory nerve. In this way their findings clearly support the classic theory of hearing in relation to the function of the round window. Because there were certain differences in their procedure from that of Hughson and Crowe they do not imply that there were any factual contradictions. The gum plugs used by Culler and his associates were placed in apposition to the round window without pressure but with sufficient contact to impede the normal oscillation and thereby impair the acuity of hearing, according to the accepted theory of cochlear function.

In a study of the threshold of the reflexes of the muscles of the middle ear with the tympanic membrane intact and also after extirpation, Lorente de Nó and Harris [26] summarize their observations as follows:

1. The threshold curve of acoustic reflexes of the muscles of the middle ear in the rabbit bears the same general configuration as the human threshold curve of audition, showing the greatest sensitivity of the hearing mechanism at around 2,000 to 4,000 cycles; but the threshold does not rise as rapidly in the high pitch region as in the human.

2. The curves of the tensor and stapedius do not coincide or run parallel but change their relationships with differences in pitch. . . . Thus, for tones up to 3,000 cycles the stapedius has a lower threshold than the tensor; between 3,000 and 8 to 10,000 both muscles have the same threshold or in some rabbits that of the tensor tympani is slightly lower; from 10,000 up the stapedius often seems to have again a lower threshold than the tensor tympani. . . .

3. After extirpation of the drum membrane there is a very nearly constant loss of sensitivity throughout the whole tone range. This loss is of the order of 20 decibels, but the sensitivity curve remains qualitatively unchanged. This indicates that the highest sensitivity in the area, 2,000 to 4,000 cycles, is not due to resonance in the drum membrane or middle ear mechanism.

Lorente de Nó [27] utilized the reflex contractions of the muscles of the middle ear for studying cochlear function. He says that the audiograms of the reflex contractions bear a close resemblance to the threshold curves for human beings as determined by Wegel. In human beings these reflex contractions of the stapedius muscle, as determined by Luscher and Kobrak, occur only with tones of higher intensity than

26. Lorente de Nó, R., and Harris, A. S.: Experimental Studies in Hearing: I. The Threshold of the Reflexes of the Muscles of the Middle Ear; II. The Hearing-Loss after Extirpation of the Tympanic Membrane, Laryngoscope **43**:315 (April) 1933.

27. Lorente de Nó, R.: The Reflex Contractions of the Muscles of the Middle Ear as a Hearing Test in Experimental Animals, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:26, 1933.

the sensation threshold. He says that when the contractions of the tensor tympani are recorded optically by means of an isometric myograph, accurate curves are obtained. The tension developed by the muscle gives a measure of the intensity of the reflex contraction.

The tensor tympani is innervated by the fifth and the stapedius by the seventh nerve, and in histologic preparations their motoneurons are easily recognized. Since these neurons have connections with the superior olivary body, they must be the motor cells of the muscles of the middle ear. In this same region is found the insensitive motor apparatus of the cochlear system. The author says:

> The reflexes of the muscles of the middle ear allow of making quantitative measurements, which were impossible to make with the acoustic sensations.
>
> The motoneurons of the tensor tympani function in the same way as the spinal motoneurons. Since the acoustic reflex has the same properties as the trigeminus tensor reflex and the spinal reflexes, it is concluded that the trains of nervous impulses set up in the cochlear paths by tones striking the ear must be of the same nature and obey the same laws as the nervous impulses conducted by the spinal and other sensory paths.

The author says that the "central cochlear mechanism makes an analysis of the tones in the same way as other sensory apparati analyze their own stimuli" and that this analysis does not take place in the cochlea, as was thought until Davis and Saul and their co-workers demonstrated this phenomenon.

Mink [28] offers an addition to his views on hearing published in 1930. At that time he described the action of the ossicles and explained how the membrane could communicate all variations in its relaxation to the stapes. In the present article he describes how sound in the form of pressure inscribes its modifications as currents of endolymph in the middle ear, these currents being similar to the inscription made on a victrola record.

Mink says that a study of comparative anatomy shows that hearing is only a modification of the tactile sense, and he therefore suggests that the auditory hair cells in the organ of Corti are brought into action, a given number at a time, by touching the membrane of Corti. This is accomplished by the elevation of the basilar membrane through movements of the endolymph. He describes a manometer which he has constructed which functions in the same way as the cochlea, but he admits that the cochlea, if compared to a manometer, should also have a scale of measurement. He points out that the fasciculi of the auditory nerve enter the cochlea through approximately four thousand canaliculi in the lower bony border of the spiral lamina. Each fasciculus in turn goes to its own external pillar (estimated at about four thousand).

28. Mink, P. J.: The Internal Ear as a Manometer, Ann. d'oto-laryng., June 1932, no. 6, p. 678.

From these facts it is suggested that each external pillar, with the part of the striated zone attached to it, the associated group of auditory cells and the supporting Deiters' cells must be regarded as an anatomic unit with separate innervation.

Mink says that if this point of view is accepted, the striated zone of the basilar membrane must be regarded as a series of "adjacent parts" to the external pillars, i. e., about four thousand. Therefore, as there are about twelve thousand auditory hairs, three of these hairs are raised at a time to touch the membrane of Corti. This accords with the arrangement of the hairs in three rows, as found in the organ of Corti.

In short, according to Mink, it seems much more reasonable to think that the cochlea acts like a manometer with a scale of 4,000 degrees than to regard it as a resonator, as described by Helmholtz.

McNally [29] presents a bibliographic review of the comparative physiology of hearing as reported previously by Parker, Kreidl, Stetter and others and concludes as follows:

It has been clearly proved that some fish can hear. The higher forms of fish are able to hear and differentiate between a number of tones and noises.

The pars inferior, consisting of the saccule and the lagena, is the part of the ear most likely concerned with hearing in fish.

The saccule has been shown to have no vestibular function in fish, frogs and a mammal, the rabbit. In pigeons it may be concerned with compensatory rotatory movements of the eyes.

In man the cochlea is an organ of hearing. The basilar membrane is the most likely resonating mechanism for resolving the complex sounds falling on the ear into their simple constituent harmonic components.

The apical part of the cochlea is concerned with the perception of tones of low frequency and the basilar part with the perception of tones of high frequency.

The resonance theory of hearing or some slight modification of it is most likely correct. It is probable that simple nerve impulses are sent to the brain from each section of the basilar membrane undergoing sympathetic vibration.

A fusion or synthesis of these impulses takes place in the acoustic analyzer, the nucleus of which is situated in the temporal lobe, the remnants of the analyses being distributed throughout the whole mass of the cortex.

To be concluded

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29. McNally, W. J.: Recent Advances in the Physiology of Hearing, Arch. Otolaryng. **19**:201 (Feb.) 1934.

Abstracts from Current Literature

Ear

TUBERCULOMA OF THE BRAIN ASSOCIATED WITH EAR DISEASE. J. P. STEWART, J. Laryng. & Otol. **49**:493 (Aug.) 1934.

Stewart reviews the literature on tuberculoma of the brain and reports a case of tuberculoma complicating tuberculous mastoiditis. The route of the infection is traced from the primary tuberculous focus in the adenoid tissue through the eustachian tube, middle ear and mastoid and, finally, to the brain tissue. Photomicrographs of these tissues are presented. A direct spread is demonstrated and not the usual spread of the infection by way of the blood stream. Diagnosis of such a growth is difficult unless localizing symptoms of pressure are present. As no such symptoms existed in this case, the diagnosis of tuberculoma was made only at operation. The prognosis of the condition is bad, patients rarely living more than one year. In the case reported treatment consisted in a decompression, which had already been performed by nature, removal of the diseased bone, the tuberculoma being left in situ, and ultraviolet irradiation. Extirpation of the tuberculoma is not recommended.

EDITOR'S ABSTRACT.

SPONTANEOUS CURE OF A PULMONARY ABSCESS SECONDARY TO MASTOIDITIS WITH SINUS THROMBOPHLEBITIS. B. WEILL-HALLÉ, RICHIER and A. ABAZA, Bull. Soc. de pédiat. de Paris **31**:337 (July 11) 1933.

A girl, 8½ years old, had chronic suppurative otitis media for two years. Mastoiditis followed by thrombophlebitis of the lateral sinus and thrombosis of the right jugular vein developed. Two weeks after she had been successfully operated on she again showed hyperpyrexia, and it was found that she had a metastatic abscess of the apex of the left lung. During the following three weeks the abscess healed. There had been no vomiting and no special treatment.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

OTOGENOUS SEPSIS IN CHILDREN. RUDOLF LEIDLER, Wien. klin. Wchnschr. **46**: 1128 (Sept. 15) 1933.

Leidler asks: What local signs betray severe complications to the physician? Because of the importance of this question for the pediatrician, the following important points are stressed:

1. The appearance of symptoms of general sepsis is of first importance in the diagnosis.

2. Special local symptoms in the ear and the surrounding area (except in the case of the existence of otitis immediately prior to the appearance of sepsis) need not be present; it is even the rule that primarily no striking signs (such as subperiosteal abscess) may be found.

3. In many cases there exist: mild tenderness over the mastoid process and moderate swelling of the soft parts, a profuse flow of pus from the ear or sudden stopping of the suppuration (especially in chronic otitis), painfulness on point pressure at the posterior rim of the mastoid process (the area of the emissarium mastoideum), painfulness at the anterior rim of the sternocleidomastoid muscle (periphlebitis) and (very infrequently) palpability of a hard tract therein (a thrombosed vein or pervascular infiltration).

4. The appearance of painful or painless swollen lymphatic glands at the angle of the jaw on the side of the otitis is a suggestive symptom, especially for bulbus thrombosis.

5. Sinus thrombosis in infants is infrequent, in contrast to a septic course without alteration in the sinus.

6. Proof of the presence of micro-organisms in the blood (especially Streptococcus haemolyticus) frequently indicates sepsis; however, their absence does not preclude sepsis. This is analogous for leukocytosis, with a shift to the left, and also for splenic enlargement.

ABT, Chicago. [AM. J. DIS. CHILD.]

TREATMENT OF MASTOIDITIS IN CHILDREN. RUDOLF LEIDLER, Wien. klin. Wchnschr. **46**:1458 (Dec. 1) 1933.

Leidler states that mastoidectomy and opening of the mastoid antrum should be performed in the following cases:

1. If symptoms of intracranial complications are present during the existence of a marked flow of pus from the middle ear.

2. If there are symptoms of a general infection accompanied by septic fever and rigor.

3. If there is subperiosteal abscess, i. e., clearly indicated swelling of the soft parts over the mastoid process (even without pain or fever).

4. If there is evidence of Bezold's mastoiditis, i. e., a swelling first noted in the neck, below and over the mastoid process. The regional lymph glands may be enlarged, obscuring the diagnosis.

5. If severe otorrhea persists for more than three weeks and is combined with pain over the mastoid process and an elevation in temperature to more than 37.5 C. (99.5 F.), in spite of rest in bed and conservative treatment.

6. If approximately after the sixth week of severe otorrhea a temperature of more than 37.5 C. continues, and also when no other symptoms, such as swelling and pain in the mastoid process, are present.

7. If in spite of conservative treatment (with several paracenteses) severe otorrhea and deafness persist for more than eight weeks, accompanied by headaches at night, because of the possibility of the existence of a mastoid abscess and the danger of chronic otitis.

8. If the high fever and the pains in the mastoid process continue unabated at the onset of otitis for more than five days, in spite of paracentesis and good drainage of pus, because of the danger of severe complications.

A good roentgenogram aids materially in making the diagnosis and establishing the indication for operation; this is true, however, only after the mastoid process is well developed. If the roentgenogram shows softening of the bone in any place, this finding will considerably further the establishment of the diagnosis.

Mastoiditis requires special treatment, namely, operation, only when there is no further hope of cure by conservative treatment of the otitis.

ABT, Chicago. [AM. J. DIS. CHILD.]

Pharynx

ASTHMATIFORM ATTACK PROVOKED BY MECHANICAL IRRITATION OF A TONSIL (A PIECE OF A GRAIN OF OATS). P. ARMAND-DELILLE and H. GAVOIS, Bull. Soc. de pédiat. de Paris **31**:150 (March 21) 1933.

A girl 2½ years old was admitted to the hospital because of severe dyspnea of the asthmatic type. On the third day after admission a grain of oats was removed from the right tonsil. The illness ran a febrile course for ten days, although the breathing, which had become considerably improved, became normal soon after the removal of the foreign body from the tonsil. Considerable inflammatory reaction of the mucous membrane was present around it, and it is considered that an extension of this process to the tracheobronchial mucosa, as well as a reflex irritation, may account for the marked respiratory distress. Examination of the blood showed no eosinophilia.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

RELATIONSHIP BETWEEN DIGESTIVE DISORDERS AND DISEASES OF THE THROAT. J. GUERRA ESTAPÉ, Pediatría españ. **21**:637 (Oct.) 1933.

The author points out the definite connection which exists between these two disorders but believes that the relationshp is characteristic for certain constitutional types. In advising tonsillectomy and adenoidectomy he is influenced not so much by the hypertrophy of these structures as by the constitutional make-up of the child and certain disabilities which have developed, such as abnormalities of respiration and phonation and recurrent infections of the nose, ears and bronchial tract. He advises against indiscriminate operative procedure in the throat.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

Larynx

MALIGNANT DISEASE OF THE LARYNX AND PHARYNX. A. ZUPPINGER AND R. STEWART-HARRISON, J. Laryng. & Otol. **49**:720 (Nov.) 1934.

Zuppinger and Stewart-Harrison present a "follow-up" investigation of a number of patients with malignant growths of the larynx and pharynx who formed the subject of a provisional report previously published in the same journal in November 1932. One hundred and fifty-nine patients were studied. Of these, 120 completed treatment. At the time of writing 27 patients were living and had been free from all disease for from two and one-half to five years after treatment. Nine who died of intercurrent disease were free from local symptoms during the first six months after treatment. Fourteen died of intercurrent disease more than one year after treatment. In 25 cases, distant metastases developed in spite of freedom from local and regional disease. Fifteen recurrences were seen, only 1 of which occurred after the first year. After an observation period of two and a quarter years the local success was 53 per cent, the relative success 26 per cent and the absolute success 17 per cent. The treatment was Coutard's protracted fractional roentgen therapy with the following slight changes from that method as previously described: The tube tension was increased from 170 to 180 kilovolts. The tube current was increased from 3 to 4 ma. The filter was increased to 1 mm. aluminum plus 2 mm. copper. The focal skin distance exceeded 70 cm. The intensity of the radiation was made 2.5 roentgens per minute measured in air. While the primary result is satisfactory, the late results leave much to be desired. The authors believe, nevertheless, that a distinct advance is obtained over surgical treatment, and they hope for still better results from improvement in technic.

EDITOR'S ABSTRACT.

LIPOMA OF THE LARYNX—INTRINSIC IN ORIGIN. H. S. BIRKETT, J. Laryng. & Otol. **49**:733 (Nov.) 1934.

Birkett describes the case of a man, aged 28, who complained of gradually increasing hoarseness and cough. Stridor was audible, but the interference with respiration was insufficient to call the extraordinary muscles of inspiration into action. Indirect laryngoscopy showed a marked swelling of the left false cord extending from slightly beyond the anterior commissure on the right to the posterior attachment. A preliminary tracheotomy was performed and the main operation undertaken by means of laryngofissure. The larynx, however, did not have to be opened since, when the skin and subcutaneous tissues were retracted on the patient's left side, a small bright yellow mass was exposed, which was easily enucleated by blunt dissection. The specimen consisted of an irregular, lobular, pedunculated mass weighing 23 Gm. The sections showed the typical fat reticular tissue of a lipoma. By indirect laryngoscopy the larynx appeared practically normal at the end of a week. This lipoma was of intrinsic origin, a rare condition, and the author was able to find only four other cases of intrinsic lipoma reported in the literature.

EDITOR'S ABSTRACT.

PARALYSIS OF THE DILATOR MUSCLES OF THE LARYNX. RAMADIER, LÉVY-DEKER and CHAUSSÉ, Rev. d'oto-neuro-opht. **11**:749 (Dec.) 1933.

Paralysis of the dilator muscles of the glottis and its differentiation from paralysis of the recurrent laryngeal nerve are discussed, and two cases are reported. In one, the paralysis was caused by neurosyphilis, and for a long time was the sole discoverable sign of syphilis, except the loss of the photomotor reflex; in the other case, the paralysis arose from syringobulbia. In both patients an emergency tracheotomy was necessary. In the first case, the suspected diagnosis of syphilis was confirmed by the subsequent development of an undoubtedly tabetic syndrome.

While it is true that undoubted cases of paralysis of the dilator muscles have occurred in typhoid fever, diphtheria, saturnism, certain peripheral lesions (cancer of the esophagus or of the thyroid body) and syringobulbia, a practical rule is the one expressed by the authors in 1922: "All paralysis of the dilators of the glottis which does not prove its origin is syphilitic," and the therapeutic corollary is to apply systematically to this cryptogenetic laryngoplegia the treatment for neurosyphilis, since this offers the only chance for cure.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL & PSYCHIAT.]

LARYNGEAL PARALYSES FOLLOWING INJECTION OF THERAPEUTIC SERUM. H. ROGER, PRÉVOT, WAHL and BRÉMOND, Rev. d'oto-neuro-opht. **12**:419 (June) 1934.

Two cases of laryngeal paralysis following the injection of antitetanic serum are reported. In the first case the paralysis involved the dilator muscles of the glottis and the soft palate and appeared seven days after the injection; serum sickness developed and was followed within a few hours by paralysis. Syphilis was definitely eliminated, and no cause other than the effect of the serum could be discovered. Nervous accidents following various forms of serum therapy are known to have occurred. The paralyses are usually located in the brachial plexus but may be of polyneuritic type; it is exceptional for the reaction to be localized in one or two cranial nerves. In the first case the lesion was assumed to be in the bulbar nuclei of the tenth and eleventh nerves or their intrabulbar fibers. In the second case the right vocal cord and the aryteno-epiglottic muscle were involved, paralysis of the right superior and inferior laryngeal branches resulting. In spite of repeated and thorough examinations, no cause other than the serum sickness could be elicited. Only two similar cases are known to the writers: one reported by Lavrand and one reported by Schauwecker.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

Nose

SARCOMA OF THE NOSE IN AN INFANT. HAROLD EDWARDS, Proc. Roy. Soc. Med. **27**:27 (Nov.) 1933.

A case of sarcoma of the nose is reported in a child aged 2 years. The patient was cured when a few months old by treatment with radium, goat serum and erysipelas and prodigiosus toxins (Coley). Eight months later a lump appeared in the submaxillary region; the subsequent history is given in the report.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

CASE OF NASO-ORBITAL MENINGOCELE.. J. SÉDAN and P. COULOMB, Ann. d'anat. **171**:804 (Oct.) 1934.

Sédan and Coulomb report this case because of the relative rarity of the condition and because of the absence of contributions relating to it in the literature during the last few years.

Van Duyse gave a complete bibliography to 1905 in the "Ophthalmological Encyclopedia" of that date. In the same year Rohmer stressed the difficulty of diagnosis in such cases. In 1906, Parsons and Coates presented a case before the Ophthalmological Society of the United Kingdom and reported later that inter-

vention had proved fatal. Peters reported a case of bilateral encephalomeningocele in 1917. Up to 1915, sixty cases had been reported. Most of the articles are not illustrated with roentgenograms. The details of the present case are given, and two roentgenograms demonstrate the condition excellently.

McKee, Montreal, Quebec, Canada. [Arch. Ophth.]

Modifications of Mariotte's Blindspot Under the Influence of Nasal Excitation. A. Magitot and A. Dubois, Bull. Soc. d'opht. de Paris, February 1934, p. 85.

Using Evan's method of angioscotometry, the authors compared the fields taken on normal persons before and after using a tampon saturated with a 10 per cent solution of procaine hydrochloride in the nasal cavities. While the results varied somewhat in each patient, in the majority there was an enlargement of the blindspot following cocainization. The enlargement was bilateral and lasted about six minutes, after which there was a return to normal. The authors submit their results in order to show how vasomotor phenomena may influence blindspot determinations. Certain operative procedures on the sinuses when retrobulbar lesions are suspected often bring relief. The authors believe that this relief may be in part due to vasomotor changes. Mayer, Chicago. [Arch. Ophth.]

Miscellaneous

Congenital Atresia of the Esophagus. L. T. Perrault and H. J. Burman, New York State J. Med. **33**:1092 (Sept. 15) 1933.

A child, 9 months of age, had an obstruction due to a "congenital membrane" 12 cm. from the alveolar margin. It was successfully treated by dilation.

Aikman, Rochester, N. Y. [Am. J. Dis. Child.]

The Operative Treatment of Facial Palsy, with Observations on the Prepared Nerve Graft and on Facial Spasm. C. Ballance, J. Laryng. & Otol. **49**:709 (Nov.) 1934.

Ballance reviews the different operative procedures employed in the treatment for facial paralysis. He then gives a detailed account of the work which led him to use an intratemporal operation in which nerve grafts are placed directly in the fallopian canal. He has carried on the experimental work largely in association with Duel. Monkeys were used. More recently, Duel has employed this method with success on patients. Degenerated nerves are preferred to fresh nerves as grafts for the reason that the degenerated nerve is composed of a series of tubes filled with fatty masses due to the breaking up of the sheaths. Through these empty tunnels the axis-cylinders slide along with great ease. Ballance describes in detail the condition of the facial medullary nucleus of a patient with facial palsy and compares it with that of the cells of the facial nucleus of one of their experimental animals. In man the operation for the cure of facial palsy is often delayed for many months or even years in the vain hope that recovery from the palsy will take place without operation. The cells of the facial medullary nucleus become atrophied. The activity of the tissue and the vascular activity are in a state of stable equilibrium. There are no degeneration processes and no associated vascular changes. This probably explains the fact that in man facial spasm following a remedial operation is not observed. Editor's Abstract.

Lymphangioma of the Neck. A. Martin and M. Lelong, Bull. Soc. de pédiat. de Paris **31**:212 (May 16) 1933.

A girl had a lymphangioma at the base of the neck on the left side, which extended retrosternally for a short distance. The sac was ruptured at operation, and a thin chocolate-colored fluid was released. About 2 cm. of the sac had

to be left out of the excision, as it was adherent to the external aspect of the internal jugular vein. This area was painted with a solution of zinc chloride. The child made a good recovery.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

THE VESTIBULAR FUNCTIONS OF THE FRONTAL LOBE WITH THE STRIATED BODY: CEREBRAL AND SUPRATENTORIAL ATAXIA. L. J. MUSKENS, Rev. d'oto-neuro-opht. **12**:1 (Jan.) 1934.

The reasons that the functions of the frontal lobe are so poorly understood are neglect of the principle of Griesinger (anatomy first, physiology next and then the clinical picture), brutal methods of intervention on the frontal lobe and the ignoring of vestibular and supravestibular disturbances, especially those occurring in an anteroposterior direction. Frontal ataxia is not a true ataxia, a tabetic dysmetria or a true incoordination but comprises the symptoms accompanying attitudes or forced movements and the concomitant oculomotor manifestations in the vertical plane.

Clinical experience teaches that every organic disease in the anterior part of the brain disturbs the function of the anterior part of the striatum and vice versa; anatomic investigation shows that the blood supply of the anterior part of the brain is such that destruction of the basal part of the frontal lobe must necessarily block certain arterial territories in the pallidum.

The following assumptions are justified: The striate body is a major center for the production of automatic movements; the globus pallidus is a supravestibular organ; the pallidum (paleostriatum) is the principal center of association and is developed concurrently with the frontal cortex; the putamen and the caudate nucleus (neostriatum) are its physiologic and histologic counterparts; there are pathways of association between the cortex and the thalamus, between the paleostriatum and the neostriatum, between the globus pallidus and the body of Luys and between the pallidum and the vertebral trunk. Consequently, the neostriatum is an organ of sensory correlation, grouping various centripetal influxes, and the paleostriatum is an organ of sensory execution.

In all cases, branches of the anterior cerebral artery terminate in the globus pallidus, and they are certainly cut or blocked during an intervention involving solely the frontal cortex. When, therefore, typical striatal symptoms appear following such intervention, they should be attributed to these inevitable striatal foci. This fact speaks against the assumption of direct relations between the cortex and the pallidum. Hoche's researches showed that pyramidal fibers terminate in the motor nuclei of the fifth, seventh, eleventh and twelfth nerves, but none have been found in those of the third, fourth and sixth nerves; Muskens remarks that the rôle of the striatum in ocular movements has been underestimated. There exists no incontestable result from experimentation, anatomic or physiologic, that obliges the acceptance of the existence of supravestibular functions in the frontal cortex.

From a clinical point of view, the reports on frontal ataxia by numerous authors are criticized. Bruns and Oppenheim first insisted on the regular occurrence of ataxic phenomena in patients with frontal tumors: instability of the body, staggering and reeling and a tendency to fall backward. The belief of Subirana that the "small step" gait is a vestibulospinal syndrome is not concurred in. The hypothesis that ataxia in cases of supratentorial tumors is caused by a "pressure cone" is not accepted, because ataxia has no localizing importance unless it appears early and because in numerous cases the pressure cone was lacking. The fact that in cases of supratentorial tumors median tumors cause ataxia oftener than lateral tumors is comprehensible if one attributes the principal rôle to the frontopontocerebellar tract or to the neostriato-olivary bundle (Muskens); the frontopontocerebellar tract probably does not exist.

When investigating the functions of the anterior part of the brain, physiologists have too little taken into account the various nuclear and fascicular complexes

and the existence of supravestibular centers in the striatum. Muskens showed in 1914 that *mouvements de ménage* persist for months and are produced only after wounds of the anterior part of the brain involving the globus pallidus. Schäfer, Sherrington and others showed that the prefrontal electrically excitable zone was without connection with the motor zone and that stronger faradic currents were necessary to stimulate the frontal cortex than for the central convolutions. Conjugate deviation of the head and eyes from stimulation of the frontal lobe can be attributed only to stimulation, by diffusion of the current, of the subjacent striated formations. In electrical stimulation of the central ganglia, *mouvements de ménage* were produced to the diseased side and were accompanied by a tendency of the patient to fall to the healthy side. One of the most constant symptoms after a deep wound of the anterior part of the brain is a tendency of the patient to fall toward the healthy side (modified rolling movement); other forced movements (*mouvements de ménage* toward the wounded side, retropulsion, propulsion and oblique gait) were also observed. Edwards and Bagg observed that the necrosis from radium was manifested only on the putamen and the caudate nucleus—never on the pallidum—and provoked trembling but not rotation or *mouvements de ménage*. These observations confirm Muskens' ideas as to the rôle of the pallidum. One of the most important causes of the confusion that existed between physiologists and clinicians was the lack of consideration of the tendency of the patient to rotate to the healthy side and of forced movements in the vertical plane after a lesion of the anterior part of the brain. In 1914, Muskens showed that following systematic hemisection of the posterior longitudinal bundle the point where direction of forced movements of rotation and *ménage* is inverted was at the posterior commissure, where the ascending fibers decussate (especially the vestibular fibers); that the posterior commissural nucleus and the commissuromedullary tract are the principal elements in locomotion and ocular movements in the horizontal plane; that *mouvements de ménage* toward the right could be provoked not only by lesion of the right posterior commissural nucleus but by that of the right globus pallidus and the pathways uniting these centers. Lasting *mouvements de ménage* were established only if the lateral part of the globus pallidus was wounded. Other lesions of the anterior part of the brain were followed only by transient *mouvements de ménage*. Analogous observations on forced movements in the frontal plane were made. The interstitial nucleus and the spinal tract direct movements of rotation toward the healthy side with a tendency to falling and the ocular deviation of Hertwig and Magendie; the nucleus is united by ascending and descending fibers with the anterior part of the globus pallidus. An extended lesion of this portion, associated or not with a lesion of the lenticular nucleus or of the putamen, is followed by a durable tendency to rotation and falling to the healthy side. The results of Morgan's researches on sections of the striatum agree with those of Muskens.

DENNIS, Colorado Springs. Colo. [ARCH. NEUROL. & PSYCHIAT.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, Dec. 3, 1934

FRANK J. NOVAK, M.D., *President, in the Chair*

A NEW APPROACH TO THE STUDY OF OTOSCLEROSIS. DR. J. GORDON WILSON.

This paper represents an anatomic study based on reconstructions of the area between the oval window and the cochlea, the seat of predilection for otosclerosis, showing the great variations in histologic structure which may persist there through life, with both age and individual variabilities.

(Slides are shown.) These slides show the diversity of the histologic structures in this part of the otic capsule and their varying size. These structures are related to a fissure within the bone of the capsule, fissula ante fenestram, best seen in the human fetus. The fissure contains connective tissue, areolar in its center, and cartilage, and has a connection with the vestibular and tympanic cavities. Later it is obliterated by bone either partly or entirely, though there are few cases even in late adult life in which a part cannot be distinguished.

The significance of this diversity in size and structure is not known. If one considers that the final goal to which the periotic capsule is directed is bone of diverse types—primary, lamellar, intrachondral (cartilage islands)—it is obvious that frequently this area does not reach fulfilment. One finds fibrous tissue, cartilage, enlarged marrow spaces and even small sclerotic masses surrounding enlarged haversian canals apparently indiscriminately disposed between the stapes and the cochlea. The age factor is not the only factor; individual differences are obvious; for instance, fibrous tissue may persist through life. The usual periotic development may be interrupted at any stage, and any part may increase in a ratio relatively greater than normal. The fissure may remain from vestibule to tympanic cavity, or may almost entirely disappear. I regard this area as in a stage of disequilibrium where development may be emphasized or delayed; and in this I believe the vascular factor plays an important part. As I have pointed out elsewhere, the principal vascular supply, in the fetus and in the adult, is chiefly from the tympanic cavity.

The significance of the fissula is not known. Perozzi's hypothesis, that it is necessary to the development of the cochlea, does not appear to be supported by embryology. Otto Mayer and others regard it as a synchondrosis, indicating an area of compensation for tension between the cochlear part in front and the vestibular part behind. Several objections may be urged against this. For instance, I find it difficult to bring the development of this fissure into line with the development of a synchondrosis; further, if it is so important to relieve tensions, it is hard to explain why the fissula should be wanting in the otic capsules of other mammals or why no trace of it can be noted in the fetuses of mammals other than man.

Several hypotheses have been advanced to account for otosclerosis, no one of which has been generally accepted. Wittmaack believes that it is due to the damming back of venous blood within the veins of this area. Here one must presume individual anatomic peculiarities, probably hereditary, which drive venous blood into the bone at the site of predilection, resulting in a porous condition.

Otto Mayer believes otosclerosis is a reaction to mechanical tensions. Because of ossification and obliteration of this part during growth the compensatory tensions through the synchondrosis are inhibited; a traumatism occurs, resulting in bone of otosclerotic type, able to meet strains.

Gray believes otosclerosis is a degenerative process affecting the whole organ of hearing, the result of an insufficient supply of blood to all the nerve structures concerned in the perception of sound. The preponderance of otosclerosis in women is due to the greater instability of their vasomotor system. The pathologic results are largely observed in the part subject to constant strain, namely, the part anterior to the stapes. He points out that often the deafness bears little relationship to the extent of the disease; it may be severe when the stapes is hardly fixed. The severity of the tinnitus bears no relationship to its duration.

Fraser assigns great importance to infections in the middle ear in those with a hereditary tendency to otosclerosis. Nager believes it is a nutritional change and would link it with Paget's and other bone diseases.

Through all these hypotheses there runs as a fundamental idea the notion of a congenital tendency. My slides show that in the area where otosclerosis occurs and nowhere else in the otic capsule there is histologic variability showing an individual factor. With this instability otosclerosis probably can arise as a resultant from many factors, especially vascular. As the arterial supply, and probably the venous drainage, comes from or goes to the middle ear, it is with the condition of these vessels that otologists should concern themselves. I am not prepared to offer a hypothesis; there are still questions unanswered. But I believe one is not justified in writing otosclerosis down as a hopeless degenerative process, but rather as a congenital weakness which succumbs to environmental factors. It is with the latter that, as clinicians, otologists may hope to deal more or less effectively.

DISCUSSION

Dr. B. J. Anson: Dr. Wilson has asked me to comment on the method used in preparing the reconstructions seen in his lantern slides. The Born wax plate method was employed—a method which, through a period of fifty years, has been found to be highly serviceable in accurately enlarging to gross dimensions the microscopic structures seen in serial sections.

By selection it is possible to stress either (1) the detailed form of a mass of tissue (as in Dr. Wilson's lantern slides showing the configuration of the sclerotic bone, young cartilage, etc.) or (2) the topographic relations of such a mass to familiar neighboring landmarks (as in the lantern slides showing the relation of the fistular area to the cochlea, stapes and tympanic cavity).

(The actual steps followed in fabricating a reconstruction were illustrated by lantern slides.)

Dr. Austin A. Hayden: I cannot add any scientific details to the splendid presentation that has been made of this subject by Dr. Wilson and Dr. Anson, but I wish to express my appreciation of the painstaking work which these gentlemen have shown to the Society. As one who is interested in the problems of progressive deafness in a very intimate way, I must say that while these studies may be very far from the relief which the deafened person wishes to obtain at once from the otolaryngologist, they are nevertheless very necessary steps toward the ultimate solution of their problem. I think the Society is to be congratulated, and Dr. Wilson and Dr. Anson should be thanked for their discussion this evening.

Dr. E. W. Hagens: I enjoyed Dr. Wilson's paper and agree with everything he said. I have seen a few of the models at the laboratory, and they are very interesting and instructive. All that Dr. Wilson said I would support from my own microscopic sections.

I wish to ask one question: What is your opinion about the foci that occur around the cochlea, around the internal auditory meatus, around the oval window and about the semicircular canals? How do those fit in with the difference in the anatomy that you find?

Dr. J. Gordon Wilson: The purpose of this paper is to emphasize that though otosclerosis is hereditary it can be influenced by environmental factors, by disease or poisons acting on this area in the fetus or after birth. Otologists need not be too pessimistic about the outcome. Since the deficiency of hearing can be hastened by many injurious bodily states, most of them vascular, otologists

should see that the children of otosclerotic parents have as little trouble as possible in the nose and nasopharynx. Here the otologists may be able to help.

I have never seen true otosclerosis around a semicircular canal such as Dr. Hayden and others have described; it probably is very rare.

OTOLOGISTS AND LEAGUES FOR THE HARD OF HEARING. DR. AUSTIN A. HAYDEN.

I think it is a fair and comprehensive statement to say that otologists and leagues for the hard of hearing are concerned in two things, in the conservation of hearing and in the rehabilitation of deafened persons. The first item is the prevention of loss in hearing, the thing that every deafened persons hopes will not befall the next generation, and, as Dr. Wilson and Dr. Anson have intimated, there are many things that can be brought into the factor of genetics or of genetics plus environment, or the other way around.

The statement has often been made that the prevention of loss in hearing begins before the subject is born and ends considerably after he has died. I believe that statement, general as it is, is literally correct.

The detection of loss in hearing is perhaps the first thing that must be considered in the prevention of hearing loss or in the conservation of hearing itself. The earliest detection is often made by the parent. Later, it is made in school, perhaps by the school nurse, the teacher or the school physician, and then by the otologist.

This brings up the question of hearing tests which one has heard very much more about since 1919, when Wendell Phillips formed the American Federation of Leagues for the Hard of Hearing, and since the audiometer tests were introduced and other tests became more or less frequent throughout the country. This is not the time or place to argue as to the relative values of hearing tests, but they should be considered in the proper place.

In the matter of the examination of temporal bones after death, it is in that tedious and expensive work that progress in the prevention of loss in hearing most likely lies, and work along the lines of which Dr. Wilson spoke will proceed.

Conservation of hearing assumes that hearing is present and that what is there is to be saved, and that is what leagues and otologists are most interested in. While there have been failures in conserving the hearing, that is, the patient's hearing has grown worse, nevertheless the history of otology as written in the last twenty-five years is one of which otologists can be very proud. It is not only a question of preserving the hearing but of preserving the vocal normality. This is particularly difficult if loss in hearing is progressive and severe.

There is also the need of preserving good health, for while bodily infections and deformities may be very light and quite infrequent, nevertheless they are present. The loss in hearing in the abnormal person has direct connection with the failure of bodily function, and the maintenance of proper hygiene in the nose and throat is brought into this picture, with the details of which you are all familiar. So much, in a very sketchy way, of the problems which the leagues and otologists are interested in.

They are also interested in rehabilitation. The old saying, "What cannot be cured must be endured," is very opportune. The question of rehabilitation is almost as broad as that of the conservation of hearing itself. First, there is a large psychologic factor which, if overlooked, leads to dire consequences. In my own family in the last twelve months there has been a suicide because of loss of hearing. That meant that this young man did not fit himself into the place he might well have taken, that his rehabilitation was incomplete. Medical skill, medical psychology and religion must be constantly used to prevent such catastrophies. The social influences of rehabilitation must be carefully carried out. These defects lead to isolation, and for this reason the segregation of these pupils in special rooms in the schools has been fought against by the leagues.

Again, the economic situation is something that challenges the best efforts of those interested in these people—their families and every one. There are niches into which deafened persons can fit as well, or perhaps better than those who have

hearing. A member of the League for the Hard of Hearing, has been fitted into the library of the American Medical Association, where she does excellent work. It is my belief that enough positions of this type can be arranged if deafened persons can be given an inside road and employers can get at the needs of these deafened persons.

Perhaps the most important thing in the conservation and rehabilitation is the matter of hearing aids. There are many of these. The most important, though it is not a hearing aid, is lip reading. It is stepping around, making the deaf person hear, with his eyes. Then there are the actual hearing aids. First of all, do not forget the human voice—the ability to speak distinctly, slowly, and with a fair degree of loudness. *That* people with hearing loss will appreciate more than any one thing. Then there are the mechanical hearing aids. The simplest one I know of is used by a music teacher, a patient of mine. She is a teacher of music at a college, and she always has a roll of music with her; she puts this up to her ear and asks any one whom she meets to speak through it. She gets along very well. Then there are electric hearing aids, those with amplification for several persons and the individual ones that are worn on the clothing or that the person puts around the head. Some of the hearing devices can be concealed completely inside the clothing. One manufacturer makes a sort of harness that fits inside the clothing, to carry the battery. Then there are the group hearing aids. I am proud to say that two members of the Society have had these devices installed in their churches for the benefit of deafened parishioners—Dr. Robert Sonnenschein and Dr. E. W. Hagens.

How does the otologist fit into this? He certainly fits into the place which Dr. Wilson exemplified for the members this evening. All through the country in every medical school a search is being made for all forms of progressive deafness, so in the matter of research the otologists stand alone in the conservation of hearing and the direct rehabilitation of hearing loss. Even in the various orthopedic measures used in the back of the throat much good has been accomplished, for the obstructive conditions there have yielded splendidly to otolaryngologists in the last several years and have been greatly alleviated and retarded. In all of these ways the otologists has been of tremendous service to deafened patients. In addition, I think perhaps more stress could be laid by him on the psychologic factor and on the factors of general health. I wish to bespeak the support and interest of all in these matters.

(A series of lantern slides was then presented.)

DISCUSSION

Dr. Walter H. Theobald: I hope that Dr. Hayden's remarks have fallen on receptive ears. It is fortunate for otologists that various leagues throughout the country and the American Federation of Leagues for the Hard of Hearing have been formed, because they are united on a basis which is entirely friendly to otologists. Fundamentally it is a matter of public health education. If otologists do not inform the people in matters of health through such channels as the leagues for the hard of hearing, the quack will soon be on the job with a large following.

The work of the otologist is not finished by merely making a diagnosis in a case of progressive deafness in which treatment is of little or no benefit. The patient should be given mental and social rehabilitation, and no one can guide the patient so intelligently in this direction as the otologist.

It has been stated that there are 10,000,000 deafened people in the United States. It is fair to estimate that 80 per cent of this number could be aided by lip reading. I believe that lip reading is one of the greatest assets to the person who is hard of hearing, and then come the mechanical aids.

I wish to relate the story of an outstanding case of lip reading in a young man who between 4 and 5 years of age had meningitis which totally destroyed his hearing. He could not hear a sound, and yet he went through school and became an expert lip reader. That young man attended the University of Chicago. He took down all his lectures by lip reading and scholastically led his class. He was so good that he was given a scholarship in one of the eastern universities and today

holds a high position as private secretary to one of the leading men in the country. He depends on lip reading entirely and gets along just as well as one with normal hearing.

Let me emphasize that it is the duty of otologists to cooperate with the leagues for the hard of hearing in the splendid work which they are carrying on against great odds, and they will in turn stimulate the interest of the public in matters relating to the prevention and cure of deafness. I am sure it is the earnest desire of all to promote this relationship.

DR. ROBERT SONNENSCHEIN: I appreciate the invitation of the chairman to speak, but the subject has been so beautifully and comprehensively covered by Dr. Hayden that there is really nothing to add.

One seems to have reached the age, however, not only of specialization but of superspecialization. Dr. Hayden mentioned the fact that among those who handle these patients are physicians and otologists. One hopes that otologists are physicians, even though not all physicians are otologists, and this emphasizes the fact that otologists must be physicians first. Merely to treat an ear as such means something but not everything. There have been gastro-enterologists who washed out the stomach of every one who appeared, no matter what the trouble. In the matter of rehabilitation the fact has been emphasized that the social and the psychologic aspects are important. Just as some patients afflicted with terrific ozena in atrophic rhinitis have almost isolated themselves because of this affliction, so the same tendency to withdraw from society is very common among deafened persons; these leagues for the hard of hearing provide stimulating social contacts for these persons, often the only contacts offered to them. The psychologic aspects were beautifully expressed by Dr. Gordon Berry in his wonderful paper, "The Psychology of Progressive Deafness," read before the Section on Laryngology, Otology and Rhinology of the American Medical Association at the meeting in Milwaukee, June 14, 1933.

The installation of aids for the deafened at Sinai Temple has been very kindly mentioned. The Temple has over 2,000 seats and at present 50 of these are wired. Any one who wishes to go there and hear the religious services or the lectures is furnished a headpiece by the usher. My friends and I have been delighted that so many members of the League for the Hard of Hearing have availed themselves of this opportunity.

All I can say is that I believe it is the duty and should be the pleasure of the profession in general to cooperate with the leagues for the hard of hearing in order to better the unlucky lot of these deafened persons.

THE APPLICATION OF LIP READING TO CASES OF PROGRESSIVE DEAFNESS. MISS GERTRUDE TORREY, Principal, Chicago School of Lip Reading (by invitation).

The importance of lip reading for the profoundly deafened is recognized by all, but the importance of lip reading for a person with a slight loss in hearing is not so readily accepted. My associates and I feel that when the deafness is progressive, lip reading should be taken up as soon as possible after diagnosis. The eyes will learn to fill in parts of a conversation which the ears may miss, and the person will feel that he is really hearing all. And as the loss in hearing becomes greater, he will not go through the period of anguish which usually accompanies deafness, for deafness will not be the unsurmountable barrier which it is to those who cannot read the lips, and he will not find it hard to make necessary adjustments. The problems caused by deafness are to a great extent psychologic and are due to a wrong mental attitude, wrong standards and unfortunate traits of personality which have developed with loss of hearing. Psychiatrists now try to train the child of preschool age in order to avoid wrong traits in later life, and we at the Chicago School of Lip Reading feel that by taking students with progressive deafness very early we can give training which will make it unlikely that they will develop any of the characteristics which are often associated with deafened persons.

Lip reading develops quickness of mind, alertness, responsiveness, self-reliance, concentration and an interest in others rather than in one's self. The lack of some or all of these qualities is the inevitable result of deafness unless some normal form of contact with others can be kept, and lip reading provides this contact. The result of thorough training in lip reading should be a very normal man or woman who is able to and does fill his place in the world satisfactorily. I know many such.

Lip reading is not so difficult to learn as many people think. It is the result of building up a new form of association—an association between the movement of the lips and their meaning similar to the association between the letters on a page and their meaning which enables one to read. It takes time to develop association, so lip reading cannot be learned in a few hours. It takes months, or even years in some cases, but lip reading is a very great help even before one becomes an expert, and within a reasonable length of time one should be able to make home and business contacts satisfactorily.

The quickest way to learn lip reading is through a combination of private lessons and class work, supplemented by group practice. This gives training in fundamentals and speed with a teacher, and training in understanding others in classes and group work.

We use a textbook, and give lessons which should be practiced at home in order to develop family understanding and cooperation.

Lip reading seems impossible, but to show how very possible it is, I have brought with me four expert lip readers. They are totally deaf with one exception, and this one can hear only with an instrument in aid of bone conduction. When you realize that these people would have to rely on writing as a means of understanding others if it were not for lip reading, you will understand what lip reading has done for them. All four are in business and lead lives commercially and socially which normally hearing persons would lead.

(Miss Torrey conversed with these experts, both facing them and having them read her lips in profile. They then conversed with each other.)

QUESTION: I wish to ask, what is the cost of lip reading instruction?

MISS TORREY: The average cost is about two dollars a lesson. It is less by the term. Private schools offer much free practice work and class work in connection with private lessons, so that by paying for one or two lessons a week students can get many hours of varied work.

DISCUSSION

DR. WALTER H. THEOBALD: I wish to thank Miss Torrey for this excellent presentation. One thing I believe impresses all, and that is the keen sense of humor which these lip readers have shown in their conversation.

One thought I wish to put to Miss Torrey is that of conservation of the voice. I think the lip reader should preserve the voice so far as possible for the purpose of vocal inflection. I know one man who realized something of the monotony of his voice and the wooden-like tone. He is an excellent lip reader, and teacher as well, but has taught himself the vibrations of different inflections of the voice by placing his hand on his chest. He thereby was able to distinguish the difference between high and low tones.

I wish also to express a word of thanks to Mr. Huth, who brought these earphones of studio type for the use of deafened persons. I think these earphones are for office use, for studio use, for listening to the radio and so on. The apparatus is intended for home use, and from the little contact I have had with it I feel that it has greater vitality of tone and greater clearness than other mechanical aids, and one does not have the static which is present so often in other instruments. I think that is absent in this apparatus. There is a dial which will amplify low tones, and when turned in the opposite direction, it will amplify the higher tones as desired.

DR. J. GORDON WILSON: I agree with Dr. Theobald that this has been an entertaining and instructive presentation. I wish to tell of an interesting demon-

stration I saw this year. In a phonetic laboratory a smoked drum was being used. The deaf person spoke into a cone attached to a stylet, and thereby his speech curve was registered on the drum under the curve of a normal voice. By comparing these he saw where he failed and thus had an ocular guide to better speech.

DR. ROBERT SONNENSCHEIN: In the apparatus which Dr. Max A. Goldstein uses in St. Louis, the teacher speaks into the apparatus, and the pupil then tries to reproduce the curve shown on the disk by reproducing the tones of the teacher's voice.

GERMAN OTOLARYNGOLOGICAL SOCIETY

Wurzburg, May 17 to 19, 1934

ZEITSCHRIFT FÜR HALS-, NASEN- UND OHRENHEILKUNDE **36**:251-453, 1934

ABSTRACTED BY ALFRED LEWY, M.D.

(Concluded from p. 239)

COURSE OF HEALING IN TRAUMATIC DISTURBANCES OF HEARING AND OF EQUILIBRIUM. DR. H. LEICHER, Frankfort-on-Main.

Observations were made on patients at intervals of from two months to three years over a period of from two to ten years.

Deafness.—The majority of patients showed no appreciable improvement. In those who did improve the improvement was in air conduction, bone conduction remaining unchanged. If traumatic deafness grows worse, a second trauma and syphilis should be excluded as possible causes.

Caloric Response.—Of 10 patients in whom the response was lost 6 showed an eventual return to normal. In 1 patient there was hyperirritability before restoration. In 1 patient first one side, then the other, became hyperirritable.

Relation Between Spontaneous Symptoms and Abnormal Reactions to Tests.—When there are abnormal reactions to tests but no spontaneous disturbances, these may develop later. In one case spontaneous symptoms remained after the test reactions became normal.

Vertigo.—There is usually a parallelism between the objective findings and the severity of the vertigo, which is important from the medicolegal standpoint. When objective evidence is not present, observation of the patient at his daily occupation may show evidence of vertigo. Even after disappearance of objective symptoms, a sudden severe recurrence of vertigo may cause a dangerous fall. On the other hand, two patients who had persistent loss of caloric reaction had no spontaneous vertigo.

HEREDITARY DEAFNESS AND THE LAW ON STERILIZATION. DR. W. ALBRECHT, Tübingen.

Hereditary deafness may be classified as follows: (1) otosclerosis, (2) the deafness of cretinism (type Siebenmann), (3) hereditary inner ear deafness (type Mondini) and (4) sporadic deaf-mutism (type Scheibe).

Otosclerosis.—In some families otosclerosis appears as a dominant characteristic, but in the large majority it is probably a recessive one of unknown type. In these cases sterilization should not be compulsory, but is advisable if the deafness is made worse by pregnancy.

Cretinism.—It is a question whether the deafness per se is hereditary or merely a part of the general condition. It is also a question whether it is due to a fault of the germ plasm or to extraneous influences.

Hereditary Inner Ear Deafness.—On the other hand, this type of deafness is due to changes in the germ plasm. It appears as a dominant. Characteristic for this type is the appearance of severe alongside moderate deafness in the family history. Sterilization is recommended in the severe cases.

Sporadic or Recessive Deaf-Mutism.—This occurs more frequently where inbreeding is common. The condition was studied in an isolated village of 440 inhabitants. The pathologic changes are in the nerve elements only. This type occurs sporadically, and the deafness is total or severe with only small tone islands. The value of sterilization for this type is questionable. There are too many unknown carriers. The best plan is to discourage consanguineous marriages. It is difficult to get true family histories. The deafness is usually attributed to injury or to infectious disease. A careful genealogical search is necessary. A general examination for cretinism and syphilis should be made.

There are approximately 45,000 cases of deafness in Germany, of which 15,000 are hereditary. Therefore the ratio of the cases of acquired to the cases of hereditary deafness is 2:1. They are very costly to the state, and I believe that sometime the start must be made to eradicate the hereditary type, but it will take many years to accomplish this.

THE BACTERICIDAL PROPERTIES OF THE CEREBROSPINAL FLUID IN RELATION TO INTRAVENOUS THERAPY. DR. F. KOCH and DR. F. LIEB.

Experiments were made with cultures of Bacillus paratyphosus, Streptococcus, Friedländer's bacillus, Staphylococcus and Bacillus coli in spinal fluid after intravenous injections of hexylamine, quinine derivatives and acridine dyes, as compared with control spinal fluid. We are convinced that hexylamine especially has bacteriostatic value, particularly if the intravenous injections are given immediately after a withdrawal of spinal fluid. Withdrawal appears to increase the permeability of the tissues through which the interchange from the blood stream to the spinal fluid takes place. In this respect the experiments differ from those previously reported, in which a negative finding was made.

BIOLOGIC EVIDENCE OF THE RELATIONSHIP BETWEEN GENUINE OZENA AND ANHIDROSIS HYPOTRICHOTICA. DR. OTTO FLEISHMANN.

I shall undertake to show by genealogical charts the relationship between anhydrosis, hypotrichosis, defective dentition and ozena. Ozena is related as defective development of ectodermal mucous glands. The phenomena cannot be explained on a basis of a monohybrid sex-linked recessive, but may be as a polyhybridism, resulting in a certain number of cases in the right combination of factors.

TONSILLECTOMY DURING THE ACUTE STAGE OF PERITONSILLAR ABSCESS. DR. TONNDORF, Dresden.

This is a report on 542 patients with peritonsillar abscess who were treated by simple incision through the anterior pillar, with perfect results in 99 per cent. Cases totaling several thousand are cited from other clinics, which are well satisfied with this technic. It is admitted that enucleation during the height of the abscess is not so serious as has generally been supposed, yet enucleation is unnecessary except in cases of sepsis and in the few cases in which the abscess fails to heal with simple incision. Enucleation is attended with greater morbidity than is incision, and one of its proponents has stated that it is contraindicated in the phlegmon stage before abscess formation.

BIOLOGIC MANAGEMENT OF TUMORS. DR. T. HÜNERMANN, Düsseldorf.

In 30 cases, the injection of extract of spleen, marrow and thymus (Fichera 365), as advised by Professor Fichera of the Italian Cancer Institute, in connection with irradiation by the Coutard method, has been tried. The substance increases histolysis, probably through the effect on the reticulo-endothelial system, acting

synergistically with the rays; therefore the dosage must be modified in each case accordingly. Further experiment must show whether this substance is best used before, during or after irradiation.

EXPERIMENTAL HISTOPATHOLOGY OF THE INNER EAR AND ITS NORMAL STRUCTURE. DR. C. F. WERNER, Hamburg.

Three things are necessary: (1) to broaden one's knowledge of the normal; (2) to improve one's histologic technic; (3) to simplify one's experimental methods.

The individual cells of the organ of Corti are well known, but not those of the maculae. (Author illustrates the results of fixation in the usual potassium bichromate-dilute solution of formaldehyde-glacial acetic acid solution, and in the same solution without the acid. He also contrasts the results of hardening at body temperature and at 1 C.) Addition of gelatin to the pyroxylin (celloidin) for embedding brings better results. Normally the cupola fills the ampulla. As it is seen after the usual preparation, shrinking has taken place. Fixation within twenty minutes after the death of an animal will show some results of the reactions of the living cells to the agents used, as the cells live that long. The experiment of Wever and Bray on action current conductivity in the cochlea and acoustic nerve of the cat may be cited in support of this length of life of the cells.

THE TENSION OF THE HUMAN BASILAR MEMBRANE. DR. BERENDES, Heidelberg.

Békesy showed on a model that waves initiated at the oval window produce whirlpools at certain points. These were at the wider end (tip) of the model, with use of the lower frequencies. With the higher frequencies they were at the basal (narrower) end. The whirls are looked on by Ranke as the stimulus to Corti's organ and are designated as a "rectifier" effect, and are dependent on the depth and width of the canal and the tension of the basilar membrane. The first two factors can be measured; measurement of the last is difficult.

I undertook to measure the tension of the first half of the basal coil in a recently obtained human specimen. A needle attached to two continuous axes, one of which had a hairspring attached, was placed on the basilar membrane on the scala tympani side. Both axes had mirrors attached, which reflected a beam of light on photographic paper on a kymograph. The varying pressure was thus registered, and a formula was calculated to indicate the tension.

The conclusion is: The rectifier theory of hearing advanced by Ranke depends on the tension of the basilar membrane. Berendes' instruments indicate a frequency of from 735 to 1,000 at the portion of the basilar membrane measured—therefore within the hearing range.

OTOSCLEROSIS AND CALLUS FORMATION: CONTRIBUTION TO THE HISTOLOGIC CONCEPTION OF OTOSCLEROSIS. DR. GREIFENSTEIN, Munich.

O. Mayer's theory of fracture lines as sites of predilection for otosclerosis is disputed by Wittmaack, Eckert-Möbius and others because of the absence of the usual callus coloring and structure in otosclerotic foci. This is true as applied to the ordinary healing process but not in pathologically inhibited healing. I have a specimen in which an otosclerotic area was cut by the chisel in an operation on the labyrinth and in which the healing process merges without distinctive margin into the otosclerotic area. (A long histologic discussion is included in the article, which will be published in greater detail later.)

RADICAL OPERATION VIA THE SURGICALLY WIDENED AUDITORY CANAL. DR. HANS HEERMANN, Essen.

An incision is made between the tragus and the helix, then subcutaneously to and beyond Henle's spine; a Panse flap is cut from the canal, the tissues are retracted, and an operation on the bone according to Zaufal is done. Only four of thirteen illustrations are published in this article.

Better access and view are claimed on account of the shallower wound cavity; there is no retro-auricular cut, and the plastic operation is done in advance. No special instruments are required. The scar in front of the auricle is invisible in six months. The exposure makes lesions in the middle fossa, especially in the tip of the pyramid, more accessible. The drawback is: A wider exposure is necessary in complications involving the posterior fossa.

OCCURRENCE AND MANAGEMENT OF OTOGENIC SEPSIS WITHOUT RECOGNIZABLE SINUS THROMBOSIS. DR. HAYMANN, Munich.

The various possibilities are: (1) direct entrance of the bacteria into the relatively unchanged blood vessels; (2) extension of the inflammation to the wall of the sinus, then entrance of the bacteria into the blood stream with or without thrombosis.

The former, i. e., entrance of the bacteria into the blood stream without thrombosis, is more dangerous. I believe that thrombus formation indicates better resistance. I advise free exposure of the sinus and puncture. If the sinus is macroscopically diseased, a wide excision should be made, followed by prolonged tamponade to promote thrombus formation. This is more easily said than done, because of the entrance of the petrosal sinus into the lateral sinus.

OTITIS MEDIA AS A FOCAL INFECTION. DR. W. KINDLER, Solingen.

This paper refers to focal infection alone, not to metastases, and deals with otogenic nephritis. In 1,000 cases of otitis media there were 2 per cent among the acute and 0.2 per cent among the chronic in which nephritis developed. The severity of the nephritis was not dependent on the severity of the otitis. In some cases it existed from the beginning; in some it appeared late, even after exenteration of the mastoid and even if this was done under local anesthesia. Aside from nephritis no other diseases were traceable to the otitis as the focus. The prognosis is favorable, the renal complication often healing before the otitis, but in the case of a recurrence of the otitis, nephritis may also recur. Otitis media as a focal infection seems to be selective for the kidney.

The bacteriology of the kidney could not be satisfactorily determined. I am not decided as to whether the renal complication is per se an indication for operation on the mastoid.

EXAMINATION OF THE HEARING THRESHOLD OF DEAF-MUTES WITH THE OTO-AUDION, AND IMPROVEMENT OF TONE AND SPEECH PERCEPTION BY ELECTRIC HEARING DEVICES. DR. WIRTH and DR. MILBERG, Heidelberg.

An oto-audion with a loud speaker attachment and an electric hearing device were put on the subject. It was easier to demonstrate hearing for simple tones, judged by the facial expression, than hearing for speech. It was found that familiar words only should be used.

In most of the apparently totally deaf patients the optimum of hearing appeared at c-3. It may have been tactile sense, as the vibrating membrane of the telephone could be palpated. In those who could hear only from the loud speaker, the hearing field could be heightened and broadened by interposition of an electric hearing device. Most of these had their optimum threshold at c-2, but some at this optimum showed no improvement. Some who had an optimum at c-3 and who could hear only a loud shout could hear at from 0.5 to 0.25 meter with the aforementioned apparatus. In several groups with better hearing than those just mentioned the threshold improvement was toward the upper part of the scale (19 of 55). In 5 the improvement was toward the lower end. Of 55 who could not hear better than 5 to 10 per cent between b-1 and g-2 (the Bezold necessary speech area) 13 could hear vowels, and 1 even words. Two that could hear 10 per cent plus could not hear vowels. Perception in this area and for syllables occurred only when the optimum threshold was between c-2 and c-3. The hearing vestige must be between c-2 and c-4 if an electric hearing device is to be of use.

Complete islands of deafness are rare. They disappear on use of electric devices. This idea may be of help in teaching deaf-mutes.

SYPHILIS AND TURNING NYSTAGMUS. DR. VEITS, Troppau.

A variation of the Buys-Fischer method is used in that the subject is turned for three minutes, until the rotatory nystagmus has ceased, and the postrotatory nystagmus is observed. Fifty syphilitic patients were examined. The average number of strokes, the duration and the frequency were all diminished as compared with the normal. No patient was examined in whom the condition was too early for a positive Wassermann reaction. Those in whom the Wassermann reaction had become negative at the time of examination showed the highest values for nystagmus. The number of strokes was diminished more than the duration. If one assumes that the quick component is more centrally located than the slow (labyrinthine) movement one may think that the lesion in these cases was in the central nervous system.

TUNING FORK OR OTO-AUDION. DR. BERNHARD LANGENBECK, Leipzig.

Their relative value for diagnosis and for the determination of hearing acuity are discussed, also the various methods of graphing the hearing field. Tuning forks are not adapted to the measuring of hearing acuity in percentages, because their decrement in various frequencies differs so much. This shortcoming is somewhat overcome by learning the decrement for each fork, but this requires considerable calculation. One may graph the hearing field by using the maximum intensity of each fork and determining the limits of the field. The Edelmann forks are suitable for this, but forks with thin tines give an overintensity on first being struck. However, neither does the oto-audion give accurate information as to the threshold of hearing. One may criticize the Bezold-Edelmann idea that all forks from the maximal intensity to the threshold of hearing can be measured on a graph of equal intervals; their calculations based on this idea must be wrong. On the whole, while graphs may be worked out from findings with tuning forks, they are not so accurate as those derived from the results on the oto-audion. . . . By my method of transposing tuning fork findings into sensation units fundamentally accurate results may be obtained. The oto-audion is preferable for detecting minimal changes in the threshold curve with least strain on the patient, and also for obtaining this information during the observation of progressive deafness, and for medicolegal purposes. This instrument will prove useful in examining the bilateral forms of deafness associated with tumors of the brain. In the examination of deaf-mutes for vestiges of hearing in the realm of speech, the forks have the advantage of greater intensity. The use of the oto-audion will be much more convenient when the storage battery can be done away with; this is cumbersome and not reliable enough in intensity to insure constancy of results. (Nothing is mentioned as to the usefulness of the forks in clinical diagnosis.)

COMPARATIVE ANATOMY OF THE CAVERNOUS TISSUE OF THE NOSE. DR. HELMUTH RICHTER, Erlangen.

Cavernous tissue is defined as highly developed venous spaces in the mucous membrane of the nose. Its function is to warm and moisten the air and regulate the amount of flow. This tissue was examined in man, the orang-utan, macacus and lemur.

Lemur (Nycticebus).—Most of the cavernous tissue is in the upper half of the septum and lateral wall. In the lower half it is best developed in the region of Jacobson's organ and lower turbinal, which is quite spiral in cross-section.

Macacus.—The cavernous tissue is abundant throughout, even on the septum. The only accessory cavity is the maxillary antrum, which shows no cavernous tissue.

Orang-Utan (Simia Satyrus).—The cavernous tissue is principally distributed on the lateral wall between the turbinals, on the turbinals, and on the septum in the anterosuperior portion, and opposite the posterior ends of the first and second ethmoturbinals (olfactory turbinals). The orang has only a maxillary sinus, which contains no cavernous tissue.

Man.—The cavernous tissue occurs only at the tuberculum septi and on the inferior and middle turbinals. According to Denker and Kahler, it does not occur in any of the sinuses. I found it in the nasal passages and around the ostia of the accessory sinuses in fetuses. I did not find it within any of the sinuses as reported in one case by Zange, and I think its occurrence there must be rare.

SOME PRACTICAL GUIDES TO ROENTGEN DIAGNOSIS. DR. BERGER, Münster.

I believe stereograms to be of practical value. The positions advised by Schüller, Mayer and Stenvers should be used, also the occipitonasal and lateral views for the sella turcica and sphenoids. The Bucky diaphragm is substituted for by narrowing the tube port. (Author shows by illustrations that shadows are produced by a single occipitonasal plate if the angle is not absolutely accurate.) This is automatically corrected in stereograms. (Author shows views of the widened sella turcica, also of the sella eroded by general intracranial pressure, which may produce hemianopia by distant lesions.) One must be careful in interpreting such pictures as indicating lesions in the sellar region.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John J. Shea, 1918 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch; 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: Sept. 14-20, 1935.

American Bronchoscopic Society

President: Dr. Henry Hall Forbes, 40 E. 61st St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Toronto, Canada. Time: June 1, 1935.

American Laryngological Association

President: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.
Place: Toronto, Canada. Time: May 29-31, 1935.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Perry G. Goldsmith, 170 St. George St., Toronto, Canada.
Secretary: Dr. R. L. Loughran, Sharon, Conn.
Place: Toronto, Canada. Time: June 3-5, 1935.

SECTIONS:

Eastern.—Chairman: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Southern.—Chairman: Dr. Beverly R. Kennon, Medical Arts Bldg., Norfolk, Va.
Middle.—Chairman: Dr. Daniel W. Layman, Medical Arts Bldg., Indianapolis.
Mid-Western.—Chairman: Dr. Edward H. Cary, Medical Arts Bldg., Dallas, Texas.
Western.—Chairman: Dr. Frank A. Burton, Bank of America Bldg., San Diego, Calif.

American Otological Society

President: Dr. S. J. Crowe, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Toronto, Canada. Time: May 27-29, 1935.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.